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EDITORIAL

IN this issue Brooks describes examples of a problem which sometimes happens to arise during the course of spinal caries. His choice falls upon those with a sequence of pleural or intrapulmonary hazards. The rarity of these particular risks is widely credited, for even a large cold abscess of the spine generally leaves the pleural barrier intact and the lung unaffected. The same is found with tuberculosis of the chest wall, whether secondary to spinal or to glandular disease; for, however extensive, it leads to pleural complications only in a small minority. Indeed, extrapleural pus in quantity may accompany a normal pleura in such patients. But once the pleural barrier is transgressed, or the lung similarly, not only are the immediate consequences apt to be severe, but the whole course and outlook are affected.

Brooks found a demonstrable paravertebral abscess in all his cases; and in three out of six this demonstration preceded the intrathoracic catastrophe by many months. All of them had already been under treatment for lengthy periods. In the remaining three a spinal lesion had not been suspected by the time the pleura or lung became involved; yet in all of these the histories suggested such a lesion, retrospectively, in that backache, unexplained fever and toxæmia, occurring in association, preceded a clinical picture of pleural effusion or a rapidly oncoming illness with copiously productive cough. As a paravertebral abscess may occasionally arise also in the course of mediastinal lymphadenitis the same risks apply with equal force on those occasions. This origin, alternative to the spine, is perhaps as frequent, though the impression which experience leaves is that here it is more often a focal glandular affection than an established abscess, which ruptures into pleura or bronchus. Though effusion or pneumonic phthisis are then attendant risks, the dire effects of the escape of the contents of an abscess of size are averted.

Reviewing his series, Brooks believes that a syndrome can be distinguished prodromal to the abscess rupturing into lung or pleura, a syndrome marked by pleural pain and an irritating unproductive cough, not infrequently of some duration before actual rupture occurs. The importance of such warning rests with the fact that at this time repeated and effective aspirations, or surgical drainage of the abscess, may avoid what is in effect a major disaster.

Once rupture has occurred, treatment is more difficult and less certain in its results. The immediate object is still to empty the abscess and so spare the pleura or lung from the severe and protracted flooding with actively infective pus which is bound to follow otherwise. Those with pleural involvement need frequent aspirations of the pyothorax, at any rate at first; for in this way the two layers of the pleura may approximate and seal by obliterative changes. An alternative would be to use posture in a way likely to drain the pleura into the main body of the abscess, and then aspirate the latter directly. Probably the best plan is to deal with the pleural collection directly to begin with, as the simplest way of being sure to give respiratory relief; and then posture and aspirate the abscess if reaccumulation is not rapid. Where the pleural collection recurs without satisfactory re-expansion of the lung, thoracoplasty should be considered if the general features permit.

Still more dangerous and more difficult to treat are those examples where rupture occurs directly into the lung. The chief hope lies with emptying the original abscess freely and then so posturing the patient that the point of drainage remains dependent to the site of the bronchial fistula, a vital detail which can be checked effectively only by radiographs taken after lipiodol injection into the abscess. To such management a few respond by closure of the fistula. If proper drainage of the abscess cannot be achieved by aspirations the alternative is evacuation after a costotransversectomy. The problem then is whether to close the abscess, reverting to further aspirations as reaccumulation occurs, or whether to rely upon tube drainage, with its attendant risks of secondary infection and all that entails for the tuberculous subject. The former seems to be the method of choice, and that most widely used at present.

It is clear, reading Brooks' paper, that time is the governing factor in all these patients if anything remedial is to be done at all. This point can hardly be emphasised enough, for otherwise a disastrous sequence of events can have no other than a fatal outcome. C. H.

GENERAL ARTICLES

PARAVERTEBRAL ABSCESS WITH RUPTURE INTO THE PLEURA OR LUNG.

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INTRODUCTION

The development of symptoms which may derive from pleurisy with effusion, empyema, or lung abscess commonly causes the patient to seek medical advice even if the onset is insidious and the subsequent course of the illness not remarkable for its acuity. The investigation of such a case which follows, including radiological and laboratory procedures, will establish the extent and nature of the thoracic lesion as a rule with considerable accuracy. However, the condition which becomes apparent at this stage is frequently so striking that the attention of those in charge of the patient tends to be focused for the most part upon the chest, whether the ætiology of the lesion disclosed is found to be pyogenic, tuberculous, or the consequence of some other infection. Furthermore, when no obvious extrathoracic cause for the observed lesion is apparent, and when, as may well happen, the intrathoracic lesion disclosed adequately accounts for the symptoms presented by the patient, the possibility that the thoracic lesion is dependent upon an underlying paravertebral abscess, in turn as a rule caused by vertebral disease, may easily be overlooked. Moreover, even if such a possibility is considered, the difficulty in establishing the complete diagnosis may be considerable, for not only may spinal caries with paravertebral abscess on occasion be itself productive of little in the way of symptoms or signs, but in the presence, for example, of an empyema or lung abscess it may often be far from simple to demonstrate the causal process with certainty in spite of adequate radiological investigation. Finally, a mediastinal abscess, usually similar in site to that caused by vertebral disease, may arise quite insidiously from diseased mediastinal glands, and may give rise to similar consequences. In such a case the localisation and dentification of the underlying disorder may be extremely difficult if not

impossible. The major diagnostic difficulty, however, is likely to arise because the above-mentioned sequence of events is not envisaged at all, for such a catastrophe has in the past usually been regarded as of extreme rarity.

Some three years ago the author met with a case of this type, and, bearing the possibility in mind, has since encountered five others. It seems, therefore, not improbable that while this syndrome is uncommon it is certainly not of rare occurrence.

Apart from its considerable intrinsic interest, more widespread recognition of the syndrome seems desirable; since, if a paravertebral abscess is known to be present, the symptoms and signs which suggest that rupture into the pleura or lung is imminent on analysis of the case records presented in this communication prove to be distinctive enough to permit the immediate application of therapy which may well prevent this disaster; while if the case is encountered after such rupture has occurred, accurate diagnosis of the true state of affairs will allow proper treatment to be instituted at an early stage, during which there may still be some hope of a successful outcome.

Case Reports.

The essential history and clinical features presented by the six cases under discussion are appended.

1. C. E. T. Welsh male, aged 28. Family history not significant.

1934 (aged 21). Erythema nodosum.

June, 1936.—Occasional slight mid-dorsal backache.

October, 1937.—Pleurisy with effusion (right). No clinical or radiological evidence of pulmonary disease. The fluid was yellow in colour, clotted on standing, and contained 55 cells per cu. mm. (mostly lymphocytes). Tubercle bacilli could not be demonstrated directly or by guinea-pig inoculation.

January to April, 1938.—Convalescence in Switzerland with complete reabsorption of the effusion, the lungs remaining normal. Work then resumed.

July, 1938.—Febrile illness with unproductive cough and an intermittent slight backache. Cause of pyrexia not ascertained in spite of full investigation at St. Mary's Hospital. The illness thereafter became increasingly severe and persistent.

October, 1938.—Pleurisy with effusion (right). Fluid was purulent, but direct examination and culture failed to show any organism. Radiological examination now disclosed an area of " infiltration " spreading out from the right hilum. Cough developed and was productive of a trace of mucoid sputum which contained no tubercle bacilli. Pyrexia and toxæmia increased. Right phrenic crush performed.

February, 1939.—Transferred to Midhurst Sanatorium, where he continued ill and in bed. Symptoms, signs and results of investigation unaltered.

March, 1939.—Cough became productive suddenly of large amounts of purulent inoffensive sputum. Tubercle bacilli could not be demonstrated in many specimens, a mixed pyogenic infection being shown in each case.

May, 1939.—Spinal caries (D. 8 and 9) with a paravertebral abscess was for the first time suspected and proved radiologically. The spine was immobilised in a plaster jacket. High pyrexia, and cough productive daily of about 5 oz. of purulent sputum continued.

July, 1939.—Surgical drainage of the paravertebral abscess was performed at St. Mary's Hospital with subsequent slight diminution in the volume of sputum expectorated and slight diminution of toxæmia.

September, 1939.—Transferred to Harefield Emergency Hospital. Partial healing of the wound had occurred, leaving two sinuses through which some drainage of the abscess, by suitable posturing in the spinal jacket, was possible. Toxæmia, however, remained severe with anorexia, productive cough, backache, increasing weakness, clubbing of fingers and toes, and albuminuria.

December, 1939, *to September*, 1941.—Albuminuria persisted and became gross in degree; œdema of the legs, and enlargement of the spleen and liver developed. Tubercle bacilli after many examinations were ultimately demonstrated in the sputum and in the pus from the abscess. The secondary infection in the abscess was found predominantly to be due to a staphylococcus, but a course of sulphathiazole proved ineffective, as did a vaccine. Despite these measures, and repeated surgical drainage, the paravertebral abscess extended and was shown by lipiodol injection to ramify extensively over the dorsal and lumbar fascial planes. The pulmonary condition deteriorated and bronchiectasis developed around the fistula into the right lower lobe, though no radiological evidence of pulmonary tuberculosis was found. Clinically and radiologically, however, the spinal caries improved, new bone formation occurred, and it became possible to remove the spinal jacket in December, 1940. Recurrent hæmoptysis, probably from granulations in the abscess, became a feature of the case in January, 1941, and the anæmia which developed contributed to the downhill course and necessitated repeated transfusions. During 1941 he continued markedly toxic with high irregular pyrexia and a cough productive of about 5 oz. of bloodstained offensive purulent sputum daily, had well advanced amyloid disease, and, since the ramifying abscess could not adequately be drained, the prognosis was regarded as hopeless. He died in September, 1941. Post-mortem examination was not obtained.

COMMENT.—It would seem possible that a late primary tuberculous infection occurred in 1934 and dissemination led to spinal caries productive of symptoms two years later. A paravertebral abscess from this source caused pleural irritation and effusion in 1937. The abscess became secondarily infected and ruptured into the pleura in 1938, and thence into the lung in March, 1939. The bronchial fistula failed to close in spite of repeated drainage of the abscess.

2. A. H. English female, aged 26. Family history not significant.

April, 1934 (aged 21).—Pleurisy (left).

December, 1935.—Mid-dorsal backache and pyrexia led to the clinical diagnosis of spinal caries, which was confirmed radiologically (D. 7 and 8), a small paravertebral abscess also being shown to be present. She was admitted to the Royal National Orthopædic Hospital and the spine was immobilised in plaster for eighteen months. She was then able to be discharged and returned to her home in May, 1937, remaining well for a year.

June, 1938.—Increasingly severe cough productive of traces of mucoid sputum developed, and to this symptom were added dyspnœa, amenorrhœa, anorexia, loss of weight, and pleuritic pain on the right side.

September, 1938.—Pulmonary tuberculosis was diagnosed on clinical and radiological grounds by her Tuberculosis Officer. While awaiting admission she apparently became much more ill.

December, 1938.—Admitted to Harefield Sanatorium, where in addition to the above symptoms she complained of backache (level of D. 7). She was extremely toxic and ill, and while in bed on absolute rest was found to have a temperature which varied between 103° and 99° F. The physical signs suggested infiltration and fibrosis to be present throughout the right lung and in the upper part of the left lung. A pleural rub was heard over the right lower lobe. The fingers showed well-marked clubbing. The sputum was purulent and occasionally bloodstained; it had gradually increased to about 4 oz. daily, and did not contain tubercle bacilli (five specimens tested). Radiological examination showed an ill-defined opacity lying posteriorly in the middle and lower zones of the right lung extending out from the mediastinum, associated with considerable pleural reaction. This opacity was continuous with the paravertebral abscess adjacent to the seventh and eighth thoracic spines. In the left lung localised infiltration was present in the upper zone. Thoracentesis at the right base was productive of bloodstained purulent fluid, which contained staphylococci, but tubercle bacilli could not be demonstrated directly or on culture. Continuous pain, increasingly voluminous, purulent and occasionally bloodstained sputum, with growing toxæmia and high pyrexia, led to her clinical condition rapidly and progressively becoming worse.

February, 1939.—Radiological examination (Fig. 1, Plate X) showed that the opacity on the right side of the thorax had increased in size, while in the upper zone a fluid level could be demonstrated. A new lesion had appeared in the middle and lower zones on the left side. The radiologist felt that "the condition was primarily extrapleural though there were probably secondary changes in the lungs".

The patient died on March 8, 1939.

Post-mortem examination showed the presence of caries involving the seventh and eighth dorsal vertebræ, associated with a very large paravertebral abscess which had ruptured through the adherent right pleura into the lower lobe of that lung. Within the upper lobe some relatively minor chronic tuberculous infiltration was present, but in the lower lobe a large abscess containing pus was present, surrounded by a zone of septic pneumonia

PLATE X

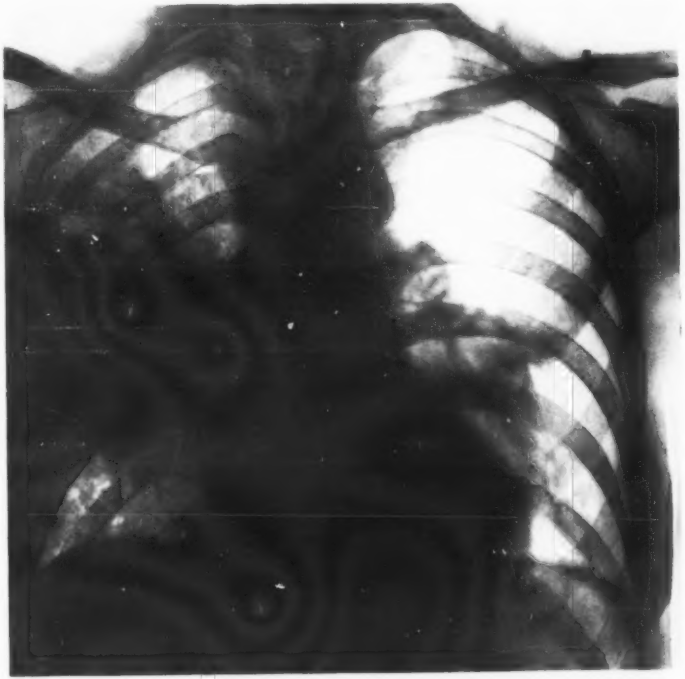

FIG. 1 (CASE 2).—SPINAL CARIES AND PARAVERTEBRAL ABSCESS, WITH RUPTURE INTO THE RIGHT LUNG.

A lung abscess has resulted toward the apex of the right lower lobe with adjacent pneumonia, and a bronchogenous spread of the suppurative process has taken place in the left lung.

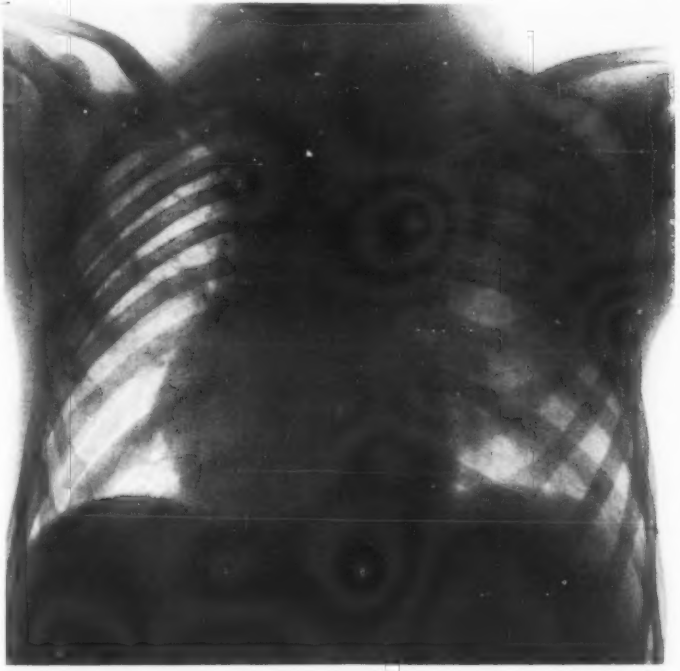

FIG. 2 (CASE 4).—SPINAL CARIES, PARAVERTEBRAL ABSCESS WITH RUPTURE INTO THE LEFT PLEURAL CAVITY.

The patient's posture was continuously such that the upper thorax was lowermost in position.

[*To face page 52.*

and bronchiectasis. In the left lung chronic and partly healed tuberculous infiltration was present in the upper lobe, while toward the lower part of this lobe multiple small abscesses and pneumonic consolidation were found. The pleural surfaces were adherent most markedly at the bases.

COMMENT.—Known tuberculosis of the spine with paravertebral abscess which had apparently responded to treatment again became active and caused pleural irritation in June, 1938. By December, 1938, the abscess had ruptured across adherent pleural surfaces into the right lung, with the formation of a lung abscess which killed the patient. The slight, mostly healed bilateral apical tuberculosis was probably the relic of the original hæmatogenous dissemination.

3. G. W. English male, aged 35. Family and previous personal history not relevant.

December, 1936.—"Influenza." Never thereafter really healthy.

July, 1937.—Persistent backache led to the diagnosis of spinal caries (D. 4, 5 and 6) with a paravertebral abscess. Tuberculous osteitis of the ribs was also found extensively to be present. He was admitted to the Brompton Hospital, where, in addition to immobilisation of the spine, surgical therapy (removal of 2nd, 3rd, 10th and 11th ribs on the right side) was necessary. Histology of the excised ribs proved the lesion to be tuberculous. He made a good recovery and a year later returned home to work, and from his own account remained well.

June, 1940.—Girdle pain felt more on the right side of the chest, weakness in both legs, and swelling of the left index finger, all at first slight but progressively increasing, forced him again to seek medical advice.

September, 1940.—He was admitted to St. Vincent's Orthopædic Hospital, toxic and pyrexial, with these same complaints now well marked in degree. There, examination showed the presence of spastic paraplegia with a level of altered sensation indicating spinal compression at D. 6. Clinically there was no evidence of pulmonary disease, and this was confirmed radiologically. Active caries of D. 4, 5 and 6 was, however, present, with slight kyphosis, and a large paravertebral abscess was shown which seemed to have pushed the pleura laterally on each side of the superior mediastinum. The lesion in the index finger proved to be tuberculous dactylitis. Mantoux reaction (P.P.D. 1st strength) positive.

October, 1940.—Immobilisation of the spine on a frame relieved the toxæmia slightly, and his general condition as well as the paraplegia improved.

January, 1941.—Clinically his condition became stationary, save that he developed a slight unproductive cough. X-ray continued to show normal lungs and a paravertebral abscess unaltered in size and position.

March, 1941.—Toxæmia increased again, as did the severity of the paraplegia. The cough, now more in evidence, continued unproductive, though a few scattered râles were audible over both lungs. X-ray showed no change in the spine, abscess, or lungs. The liver became enlarged and

palpable. In view of the continued cough the possibility of perforation was envisaged, and therefore the vertebral pedicle (D. 6) was drilled and the paravertebral abscess drained through a small incision. Culture of the foul-smelling pus which was evacuated failed to demonstrate tubercle bacilli, the infection being mixed and including hæmolytic streptococci, fusiform bacteria, and Gram-positive and Gram-negative bacilli. Clinically and radiologically his condition remained essentially unaltered, save that the spasticity was temporarily a little alleviated by the operation. The unproductive cough continued.

April, 1941.—On April 3 the cough increased in frequency but remained dry, and for the first time he became dyspnœic. Clinically and radiologically, however, no change in his condition could be demonstrated. On April 5 the cough became productive of about 3 oz. of offensive purulent sputum. Coarse râles were audible at all areas over both lungs, and the patient in fact complained of a " bubbling sensation " in the chest. The sinus to the paravertebral abscess was opened and the abscess widely drained. It became obvious at operation that a large broncho-pleural fistula was present. Clinically and radiologically by the next day extensive bronchopneumonia was demonstrated. He died on April 7. Post-mortem examination was not obtainable.

COMMENT.—In this case disseminated tuberculosis originally responding to treatment again became active. The paravertebral abscess gave rise to pleural irritation in January, 1941, and in spite of drainage in March, ruptured into the lung in April, 1941. A widespread acute bronchopneumonia was rapidly fatal.

4. R. H. English male, aged 6. The patient's father had a chronic cough but refused examination. Personal past history not relevant.

January, 1939.—Experienced a severe fall on to his back.

September, 1939.—His mother noticed a deformity of his spine at the shoulder level, and he was admitted to Harefield Emergency Hospital. Clinically, the patient, a poorly nourished boy, showed the signs of Pott's disease with marked angular kyphosis of the upper dorsal spine. A small fluctuant swelling presented posteriorly to the left of D. 4. X-ray revealed caries with involvement of D. 3 and collapse of D. 4 and D. 5, together with a paravertebral abscess which was most obvious on the left side of the superior mediastinum. His spine was immobilised in extension on a frame. Mantoux reaction positive (P.P.F. 1st strength).

January, 1940.—Pain was felt under the left clavicle following an unproductive cough, both of which symptoms lasted only for a few days. The physical signs suggested the presence of fluid over the left upper lobe, which, since he was constantly on a spinal frame, was the most dependent part of the hemithorax. The liver was palpable. X-ray confirmed the signs, showing that the paravertebral abscess had ruptured into the pleura, partially collapsing the left upper lobe and separating it along the interlobar fissure from the lower lobe (Fig. 2, Plate X). Radiologically the spine had improved as compared with the condition shown in September, 1939.

Since the pleura seemed quite free, conservative treatment was continued, and he remained on the frame until August, 1941. During this time he steadily improved. The kyphosis gradually became less and the caries slowly healed. Some absorption and calcification occurred in the paravertebral abscess and in its pleural extension. Calcification also became apparent in a left paratracheal gland.

It now seems possible that under close observation recovery may occur and the lesions may proceed to healing with little permanent functional impairment.

COMMENT.—In view of the presence of a free pleura over the left lung, and because healing of all the lesions seemed to be taking place with conservative therapy, it was felt at the time unwise to insist upon more active measures for the paravertebral abscess, in spite of the risks this course implied.

5. C. C. English male, aged 24. The patient came of agricultural stock and had lived all his boyhood in the country. Personal past history contained no significant illness.

June, 1935.—The patient complained of intermittent backache which soon became almost continuous between the scapulæ. At the same time he lost weight, felt unwell and became unduly easily tired. He remained unwell throughout the summer and autumn.

November, 1935.—A severe febrile illness with an abrupt onset developed and was characterised by cough productive of small amounts of mucoid sputum, pain in the left side, and marked toxæmia. The physical signs suggested the presence of fluid in the left pleural cavity, and a diagnosis of pneumonia with synpneumonic empyema was made. Thoracentesis revealed the presence of relatively thick pus which was sterile on culture. A rib resection was performed and the empyema drained. A sinus formed which closed after four months; the patient, however, remained unwell and subject to irregular pyrexia and a productive cough.

May, 1936.—A fluctuant swelling appeared on the anterior surface of the left chest. Clinical and radiological evidence of chronic empyema was found. Sputum and pus from the chest were examined many times for tubercle bacilli, with negative results. He was then transferred to a sanatorium where X-ray confirmed the presence of a chronic empyema on the left side, but no lesion was demonstrated in either lung.

April, 1937.—The chronic empyema was drained again, and again a sinus resulted which healed in October. His general condition improved, though the cough and backache continued.

June, 1938.—A fluctuant swelling appeared between the left scapula and the spine. Clinical and radiological investigation showed that spinal caries (D. 4, 5, 6, 7 and 8) and a paravertebral abscess were present in addition to the chronic empyema. The patient was immobilised for some six months in a spinal jacket.

September, 1938.—The empyema was redrained through the first incision,

and posteriorly drainage of the paravertebral abscess was also performed. A mixed infection was found in each site; tubercle bacilli, however, could not be demonstrated, though the condition was regarded as tuberculous. Following this occasion the sinuses remained open and have so persisted. The patient continued chronically ill in sanatorium.

October, 1939.—Weakness in the legs became noticeable, and paraplegia with incontinence of urine and fæces rapidly developed. Laminectomy (D. 4, 5, 6, 7 and 8) was undertaken. The dorsal dura was found to be greatly thickened and surrounded by extensive granulation tissue at the level of D. 5. An intradural abscess below this level was opened and drained. His condition was not improved, and, though some power remained in the extensors of the legs, flexor spasms became increasingly powerful, resulting ultimately in paraplegia in flexion with nearly complete loss of sensation below the level of D. 8. Incontinence continued and an ascending urinary infection (*B. coli* and *B. proteus*) became established. Steady slow deterioration continued for the next eight months. He was transferred to Harefield Emergency Hospital in August, 1940, where I saw him for the first time.

There were then three sinuses, discharging thick pus, leading to a large chronic left empyema and paravertebral abscess. He was very toxic; amyloid disease seemed probably to be present since the spleen and liver were enlarged, and paraplegia in flexion made nursing a difficult problem. Radiological examination showed little or no active spinal disease, and confirmed the previous conclusions in regard to the chest, while lipiodol injected into the paravertebral abscess tracked into the empyema. Several specimens of muco-purulent sputum contained no tubercle bacilli on direct examination and on culture. Examinations of pus from the various sinuses were similarly negative. Neurological and orthopædic consultations were held and it was agreed that at this stage surgical intervention could not appreciably help.

December, 1940.—An increase in pyrexia and toxæmia coincided with the development of a fluctuant swelling on the right side of the posterior surface of the neck (C. 6 and 7 and D. 1). X-ray of the cervical spine was normal. Pus from the abscess contained sulphur-yellow granules and culture showed the infection to be an anaerobic actinomycosis. Similar culture from the empyema showed the same organism to be present. Blood culture was negative. In ten days he was given some 30 grams of sulphapyridine by mouth, and the abscess was drained and packed with gauze impregnated with sulphapyridine. The wound, however, broke down, and a large sinus resulted. His cough and sputum increased. Evidence of a primary focus of actinomycosis could not be found.

February, 1941.—Râles became audible posteriorly over the right upper lobe, and X-ray confirmed the presence of an ill-defined area of consolidation and early abscess formation in the right upper lobe with pleural involvement at the right apex. Sputum, which was now copious and purulent, contained neither actinomycosis nor tubercle bacilli.

He was transferred to Billericay Sanatorium in March, 1941.

Comment.—The first symptoms were suggestive of vertebral disease, and the subsequent acute illness was more probably rupture of a para-

PLATE XI

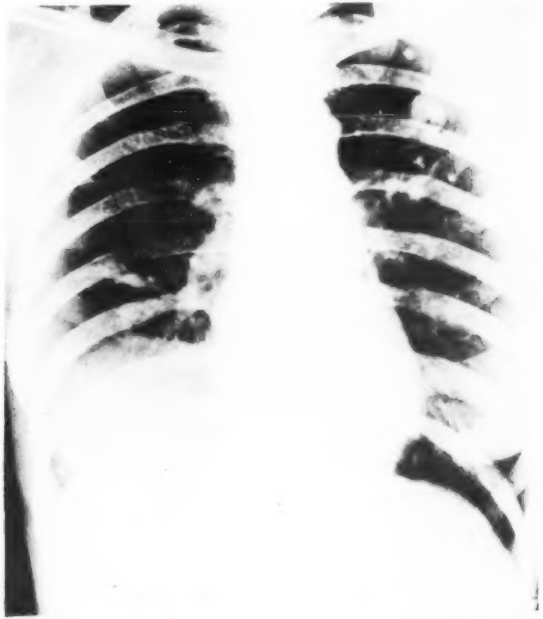

FIG. 3 (CASE 6).—ANTEROPOSTERIOR RADIOGRAM SHOWING BILATERAL PARTLY CALCIFIED INFILTRATION, AND AT THE RIGHT BASE THE PLEURAL EXTENSION OF THE PARAVERTEBRAL ABSCESS.

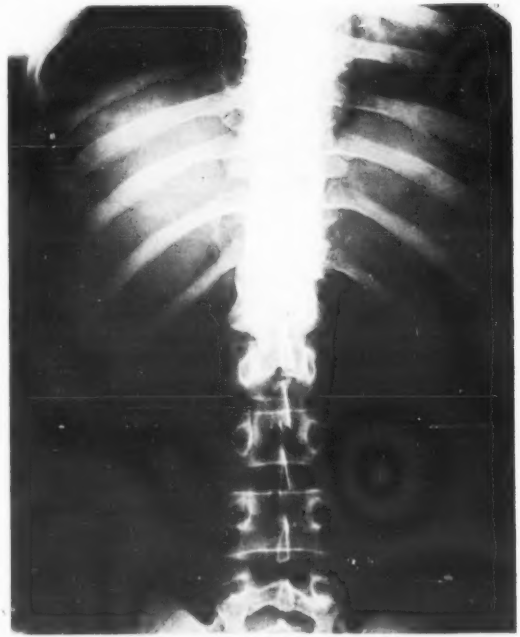

FIG. 4 (CASE 6).—SPINAL CARIES WITH A PARAVERTEBRAL ABSCESS COMMUNICATING BY A SINUS INTO THE PLEURA ON THE RIGHT SIDE.

Both the abscess and empyema show early calcification.

[*To face page 57.*

vertebral abscess into the pleura than pneumonia with synpneumonic empyema—as the symptoms and nature of the fluid show. The spinal and thoracic lesions persisted and slowly extended, with widespread abscess and sinus formation about the thorax. The lung on the right side became involved, possibly by direct extension after nearly six years' illness. The site of entry of actinomycosis remains a matter of conjecture.

6. S. E. English female, aged 28. Family history not significant. No previous serious illness.

1938.—Backache confined to the upper lumbar region developed. It was at first intermittent, but later became continuous, and was diagnosed and treated as fibrositis without success.

June, 1939.—"Pneumonia and pleurisy" (right).

June, 1941.—Continued backache, malaise and pyrexia led to the diagnosis of spinal caries, and she was admitted to St. Vincent's Hospital. Clinically some tenderness on pressure was obtainable over D. 11, D. 12 and L. 1. Adjacent and to the right of the spine from D. 7 to L. 1 movement was diminished, percussion note impaired, and the air entry was poor.

Radiological investigation showed spinal caries involving all the vertebræ from D. 7 to L. 1, D. 12 being most extensively affected. A paravertebral abscess extended from D. 11 to L. 1. At the base of the right lung a rounded opaque shadow, based on the diaphragm with a clear-cut upper margin, was present and was shown by a lateral film to lie posteriorly. In the right lung calcified infiltration was present in the upper zone, and more recent infiltration in the lower zone. The pleura was thickened in the right axilla. In the left lung slight infiltration of some standing was present in the upper and middle zones (Fig. 3, Plate XI). The patient was occasionally pyrexial while at rest in bed, but had no other symptoms. The blood sedimentation rate was 16 mm. in 200 mm. in one hour. Thoracentesis resulted in pus being extracted from the pleural space, and in this pus tubercle bacilli in small numbers were found on direct examination and on culture. It was at this stage thought probable that an independent localised tuberculous empyema was complicating the clinical picture.

July, 1941.—She was placed on a spinal frame, with relief of the backache and subsidence in the pyrexia, and was transferred to the Thoracic Unit at Harefield Emergency Hospital.

Further investigation there, including tomography, showed that the empyema was in fact in communication through a sinus with the paravertebral abscess. Calcification was shown to be taking place in the walls of both (Fig. 4, Plate XI).

It was decided to continue therapy on the spinal frame and to keep the empyema as nearly empty as was possible by frequent thoracentesis, but should this prove inadequate to drain the empyema and paravertebral abscess surgically.

Thoracentesis, productive of from 5 to 10 oz. of pus from the empyema, at weekly intervals during the next three months proved to be successful,

and she made uninterrupted progress. Tubercle bacilli remained present in the pus in small numbers. She continued symptom-free and apyrexial, and the sedimentation rate fell to 10 mm. in 200 in one hour. Radiological control of the state of the vertebræ, the empyema and abscess, also showed satisfactory progress. In order to diminish the risk of sinus formation which frequent thoracentesis entails, local anæsthesia had not been used.

COMMENT.—The patient apparently had caries with a paravertebral abscess which ruptured in 1939 into the pleura a year after the onset of symptoms. The symptoms subsided, only to become evident again when renewed activity of the vertebral lesion took place in 1941. Tomography revealed the communication between the paravertebral abscess and its pleural extension, and repeated aspiration has so far proved to be satisfactory therapy.

Discussion.

Of the cases presented, four were male and two female; while at the time of rupture of the paravertebral abscess into the pleura or lung their ages varied from 6 to 35 years, with an average of 24·5 years. In four of these patients this event occurred during the third decade of their life. The onset of symptoms suggestive of spinal caries approximated to an average age of 21 years.

In every case of the series spinal caries with paravertebral abscess was demonstrated either during life or at post-mortem examination to be present. In three cases (Nos. 2, 3 and 4) this demonstration antedated the rupture of the abscess into the pleura or lung by thirty, forty-five and five months respectively. Of these three cases, when perforation occurred, Nos. 3 and 4 had been in hospital for seven and five months respectively, receiving therapy (immobilisation) for their known spinal lesion; while case 2 had received eighteen months' treatment of this kind in hospital, had been discharged to her home, and had then remained, as far as is known, quite well for a further thirteen months. In the remaining three cases (Nos. 1, 5 and 6) an underlying spinal lesion was unsuspected when rupture of the paravertebral abscess into the pleura or lung occurred. Examination of the histories of these cases reveals that active spinal caries productive of symptoms probably antedated the perforation respectively by twenty-eight, five and twelve months. The duration of the interim, though less, is probably not significantly different, therefore, between the undiagnosed and the diagnosed (and treated) cases.

Tubercle bacilli were found in specimens from these cases during life in Nos. 1 and 6, while undoubted tuberculosis was demonstrated at post-mortem in case 2. Actinomycosis was demonstrated during life in case 5. It is noteworthy that the correct and complete diagnosis was made im-

mediately perforation occurred in cases 3 and 4—indeed, the catastrophe was anticipated, but not prevented by surgical intervention, in the former instance; while in case 2, although during life the complete diagnosis was not made, the possibility was considered prior to the patient's death. In the remainder (Nos. 1, 5 and 6), the full diagnosis was not made until long after perforation of the abscess into the pleura or lung had occurred.

During the period of observation the consequences of rupture into the lung proved fatal in three cases. In case 1 death occurred from pulmonary suppuration, exhaustion, and amyloid disease thirty-six months from the date of rupture, while in case 2 the interval was approximately eight months. In case 3 an acute fulminating bronchopneumonia killed the patient in three days from the time of rupture of the paravertebral abscess into the lung. Case 5 is, as far as we have been able to ascertain, unique in the literature of the subject, since no previous instance has been recorded in which an actinomycotic lesion of the spine gave rise to this series of events. Cope[1] quotes several instances in which vertebral lesions due to actinomycosis have been recorded, and discusses the possible mechanism whereby the organism reaches the spine, concluding that direct spread from the œsophagus, without necessarily leaving gross traces of œsophageal disease, is a possibility. In addition, a hæmatogenous spread from a primary infection, analogous to the accepted method of transmission in tuberculosis, may occur. In this patient, at the time of his discharge from hospital, extension of the suppurative process had occurred within the right lung and the prognosis had become hopeless.

In case 3 clinical evidence was obtained of independent hæmatogenous tuberculous lesions, while in cases 2 and 6 pulmonary tuberculosis, possibly of hæmatogenous origin, was found also to be present. In case 4 a primary complex was identified within the lung.

Amyloid disease was shown to be present in the first case, and was almost certainly present in case 5 before he left hospital. Paraplegic symptoms and signs were present before the rupture occurred in case 3 and developed subsequently in case 5. Secondary infection of the paravertebral abscess had occurred prior to rupture in case 3 and possibly antedated the catastrophe in several others.

The skin sensitivity of these cases to tuberculin would have been of interest, and it is regretted that the Mantoux test was only carried out in two (Nos. 3 and 4). In both these cases the reaction was positive in high dilution.

The rupture of a paravertebral abscess into adjacent mediastinal structures has previously been described on several occasions. In the recent literature, for example, Brand[2] quotes a case in which communication became established with an œsophageal diverticulum. The formation of a

fistula from the abscess into a bronchus has been noted in individual cases by Imbach[3] and also by de Lucchi.[4] Similarly perforation of a paravertebral abscess into the pleura has been described by Emrolaev.[5] These reports are for the most part very recent and concern isolated instances of the catastrophe, which usually had fatal consequences. Older reports are similarly limited. In the English literature no recent reference has been discovered, except that of Starkie,[6] who showed a case at the Oxford meeting of the Tuberculosis Association in 1938 in which rupture of a paravertebral abscess into the pleura occurred. He has since recently shown three cases of this accident, in each of which some degree of recovery eventually took place. It seems probable that, with the small available number of cases, considerable variation must be expected to be found in the course of the illness; and it is perhaps significant that in this series the early consequences of rupture into the pleura are considerably less severe than those in which a bronchial fistula develops. Study of these case records leads to the conclusion that a definite prodromal syndrome occurs before rupture actually takes place. In every case pleural pain, usually in addition to an irritating non-productive cough, antedated the rupture into the pleura or lung, frequently by an appreciable interval of time. The increased pressure caused by coughing may, of course, be a factor leading to perforation. Since the consequences of the perforation are not infrequently grave in the extreme, the significance of such symptoms cannot be overestimated, for it seems probable from Starkie's and, to a less extent, our experience that efficient emptying of the abscess, either by repeated frequent aspiration or—if this should prove in the slightest degree inadequate to alleviate symptoms of pleural irritation—by free surgical drainage, may prevent the impending catastrophe. It is urged, therefore, that patients in whom spinal caries and paravertebral abscess are known to be present should be specially watched with this possibility in mind; and in this connection it is noteworthy that tomography greatly facilitates accurate localisation and observation of spinal caries with or without paravertebral abscess. Its use in this field has been stressed by Lamy, Bourgeois and Thiel,[7] and in our experience the method has proved a valuable additional aid in diagnosis.

Those cases which are seen after rupture into the pleura or lung has taken place need, first, complete diagnosis; and if the possibility of this train of events occurring is borne in mind, it may be possible in some to achieve therapeutic success. It would seem desirable in general when no bronchial fistula is present to attempt completely to empty the pleural cavity and the abscess and to maintain this state of affairs by frequent aspiration, in the hope that an obliterative pleurisy will take place and will coincide with arrest of the spinal lesion. The latter is more likely to be

secured if long-continued immobilisation on a frame is insisted upon. In some cases thoracoplasty may be of value.

When a rupture into the lung has occurred—and this seems more likely to happen when the pleural surfaces are already adherent—the patient's condition is desperate, but it is perhaps of significance that bronchogenic tuberculosis does not seem often to follow. Our series would indeed suggest that the course is more likely to be either an acute suppurative process within the lung, such as is typified in cases 2 and 3, or else more chronic suppuration with the production of bronchiectasis, as was shown in case 1. In our opinion it is desirable, when a bronchial fistula is present, to drain the abscess completely and freely, modifying posture so that this drainage remains dependent and the bronchial fistula, as previously shown by lipiodol injection, maintained well above the bulk of the abscess. In this way in some cases healing of the fistula may take place. In all these cases, too, concurrent therapy by immobilisation of the initial spinal lesion is essential. Whether the train of events discussed arises consecutively to secondary infection in the paravertebral abscess, or whether it derives from the slower consequences of the original tuberculous (or actinomycotic) infection, is uncertain.

It is a pleasure to record my indebtedness to the Medical Superintendent of Harefield Emergency Hospital, Dr. A. Hope Gosse, Mr. D. McCrae Aitken and Mr. J. S. Batchelor, under whose care several of these patients were for part of the period covered by this investigation.

Summary.

Six cases are described in which a paravertebral abscess, dependent upon spinal caries, ruptured into the pleura or lung. Five of these cases were due to tuberculosis and one to actinomycosis.

An analysis is made of the clinical course followed by these patients. It is shown that prodromal symptoms suggestive of rupture of the abscess into the pleura or lung were manifested in each instance, and were present for a sufficient length of time to permit of therapy, which might on occasion prevent that catastrophe. Diagnosis, prognosis and therapy are discussed.

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THE EFFECTS AND MANAGEMENT OF TUBES USED TO DRAIN THE PLEURAL CAVITY

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In the last twenty years the difficulties of treating Acute Empyema have been resolved into two main problems. The first is how to save the patient's life, and the second how to achieve normal function of the cardio-respiratory system at the end of treatment. Stated in another way, the second problem is how to prevent the harmful effects of chronic empyema. The limelight of surgical interest has been focused on the first, and in particular upon the indications for drainage and the operative technique. The finer points of the second have not aroused the attention they merit.

The purpose of the present communication is to discuss some of the effects of pleural drainage and the practical points which are apposite to the management of a tube inserted into the pleural cavity.

Empyema compared with Simple Abscess.—Acute Empyema is a complication of inflammation in the lung or another part of the body; its reaction to drainage is particular.

Drainage of a simple abscess reduces tension in the surrounding tissues, encourages protective hyperæmia and hastens repair. The result is the same in an empyema *provided* the primary focus has resolved and pleural suppuration has been localised by adhesions. The difference lies in the fact that the presence of pus is the signal to operate upon the abscess but not the empyema. In the latter the dangers of open pyopneumothorax outweigh the harmful effects of pus in the pleural cavity, and the time for drainage is dictated by extra- as well as intra-pleural circumstances.

The site chosen for drainage of a simple abscess is predetermined by nature; cervical abscess, for instance, is incised where it points, and healing occurs before the tube has done any harm. In contrast an empyema is drained at a site chosen by the surgeon, and does not resolve before the tube has become a septic foreign body as well as a drainage channel.

An abscess whose walls are equally resilient obliterates concentrically. An empyema has walls of different consistency; the parietes are rigid and unyielding, the lung is mobile and elastic, the diaphragm muscular and active, and so the cavity is never spherical and resolves asymmetrically.

For these reasons dependent drainage is a *sine qua non* and the position

of the tube requires constant attention to meet the ever-changing anatomy of the cavity.

THE EFFECTS OF DRAINING AN EMPYEMA WITH A TUBE.—The immediate effects of drainage at the time of election are to relieve tension, to diminish toxæmia, and to allow the lung to begin re-expanding.

The later effects become significant after a few days; organisms from without then gain access to the empyema cavity by way of the tube. If a tube be introduced into a sterile pleura, infection inevitably makes its appearance after a few days; in the case of an empyema the presence of new organisms can be proved by making cultures before and after drainage. As a general rule the clinical condition of the patient is not prejudiced, because the defences of the pleura are already mobilised; but inflammation is prolonged and repair set back by the addition of new and possibly virulent organisms. To offset this danger two precautions are imperative: superinfection must be minimised by taking scrupulous care not to transfer organisms from one patient to another when the dressings are changed, and the tube must be an efficient drain and not merely a foreign body in the chest wall.

An empyema which has been drained should be regarded as an open septic wound, and inaccurate management of the tube is likely to convert a healing lesion into a chronic and disabling deformity.

When the reaction to superinfection subsides, resolution proceeds and the cavity obliterates by fusion of the granulations on the visceral and parietal pleuræ which are approximated. If the tube is taken out when the cavity has closed, the sinus in the chest wall heals without ado and the patient is cured. If it is removed when a small pleural pocket is still extant, one of two complications is probable. Either the sinus heals completely and a closed chronic empyema forms, or it becomes blocked for a few days and then breaks down and discharges pus intermittently. The former of these probabilities is the more dangerous because the inflammation is concealed and advances insidiously. A closed chronic empyema may persist for many years and conduce to ill-health and to rigid hemithorax, or the pus may rupture into the bronchial tree and engender pulmonary suppuration. In either event the patient suffers not only from the noxious effects of toxæmia, but also from gross deformity of the chest wall, the pleural cavity and the lung, and the ultimate mortality is high. The patient who exhibits a discharging sinus, after premature removal of the tube, is more fortunate, for he at least is likely to question the exactness of the cure.

The treatment of both these complications is early redrainage. If a sinus persists, it should be dilated gradually by introducing a laminaria tent for twenty-four hours so that the tube can be reinserted. Rapid

dilatation of a chest wall sinus, by finger or bougie, is open to two important risks. The first is transient septicæmia with pyrexia and malaise (comparable to that of " catheter fever ") and the second is air embolism. The initial signs of cerebral abscess are sometimes manifest after rapid dilatation. The laminaria tent must be fixed to the skin by a stout piece of silk, otherwise it will be sucked into the pleural cavity, and, being slippery, is difficult to fish out.

Superinfection is modified by the relative habits of the organisms concerned. The majority of pyogenic organisms can flourish in harmony together; some primary invaders, however, such as the tubercle bacillus and the streptothrix of actinomycosis, prefer a solitary existence and are prone to disappear from the pus when contaminated by organisms from the lung or the tube. Three practical points are pertinent to this fact. Many chronic empyemata are primarily due to one of these specific organisms, and, if the culprit is not isolated before drainage, it may only be found later on by doing a biopsy and removing a piece of the wall of the cavity for culture and section. In the second place, patients with mixed infections (*i.e.*, pyogenic organisms with tuberculosis or actinomycosis) are often erroneously assumed to have mastered the specific component of the infection because the organisms are no longer demonstrable in the pus. Finally, the onset of amyloid disease in a patient suffering from chronic empyema is a strong indication that the underlying lesion is tuberculosis or actinomycosis.

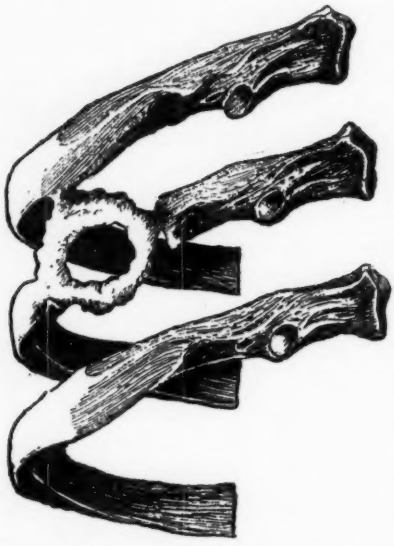

FIG. 2.—RIBS SHOWING BONE REGENERATED FROM THE PERIOSTEUM AROUND AN EMPYEMA TUBE.

In long-standing cases Bacillus pyocyaneus often makes its appearance and is evident because the pus is bright green in colour. Its tendency to spread from patient to patient is a constant reminder of the need for aseptic technique when the dressings are changed.

The primary invaders may, therefore, continue to predominate in the pus or be replaced by new organisms from without, and the organisms in the discharge may be different from those in the granulation tissue.

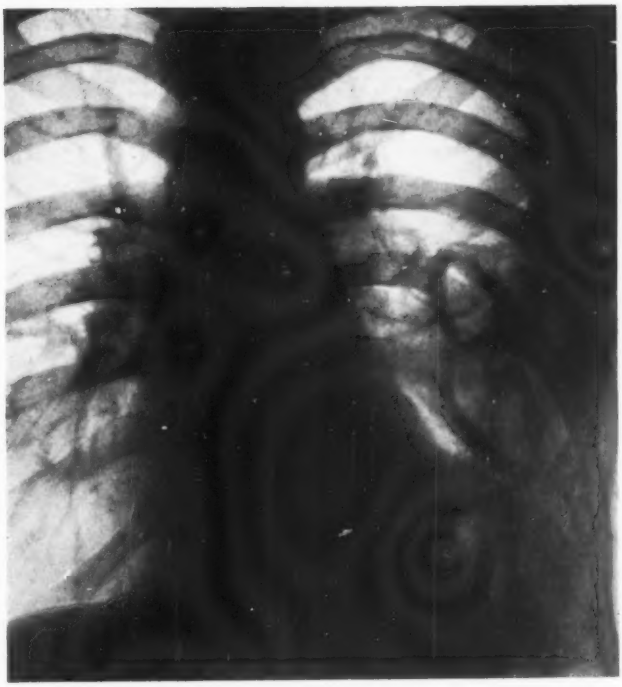

FIG. 1.—X-RAY SHOWING REGENERATION OF TWO RIBS RESECTED FOR DRAINAGE OF A CHRONIC EMPYEMA.
The operations were done at different times.

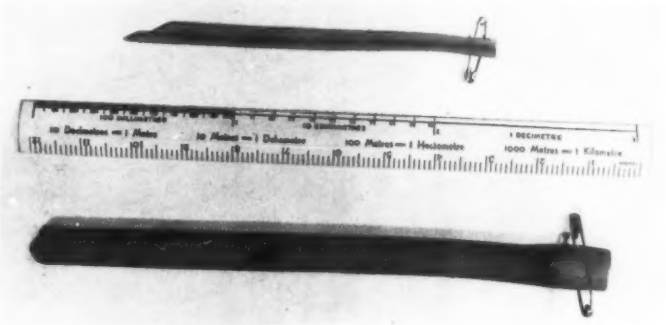

FIG. 9.—TWO EXTREME EXAMPLES OF INEFFICIENT DRAINAGE.

The patients were admitted with chronic empyemata, and in each case the condition was directly caused by the method of drainage. Corrugated rubber is never adequate to drain an empyema; it merely acts as a foreign body and aggravates pleural suppuration and infection of the chest wall.

In the second case the tube was much too large and much too long. It is obvious that as it sticks up into the empyema cavity the part above the side hole serves no useful purpose; if the side hole becomes blocked by granulation tissue, which is a common event, then the main lumen is also obstructed.

Both these patients got well as soon as drainage was corrected.

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If an empyema is slow in resolving the tube produces local changes in the chest wall. Bone regenerates from the periosteum of the rib which has been resected and forms a ring round the tube. This may be of no significance and does not often block the tube by impinging upon it, but the new bone can encroach upon the adjacent intercostal bundles and cause intercostal neuralgia, or, in later years, be mistaken in an X-ray for a pulmonary cavity. (Fig. 1, Plate XII, and Fig. 2.)

The tube track becomes rigid and fibrous with the passage of time, and eventually epithelium grows in from the surface and forms a continuous lining. This is important practically, for, whereas in acute empyema the

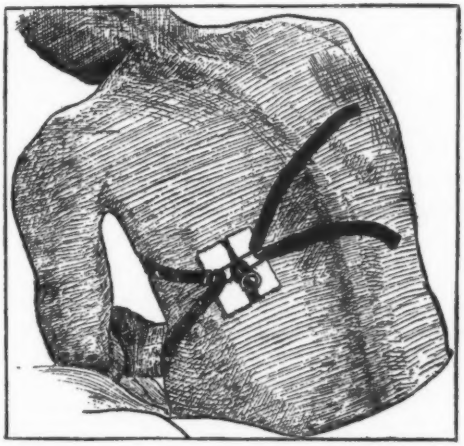

FIG. 3.—THE BEST WAY TO FIX A TUBE IS SHOWN IN THIS DIAGRAM.

It is transfixed by a safety-pin and held by two long pieces of narrow adhesive tape. If the patient is being treated by "open" drainage, the tube should be cut off ½ inch beyond the pin.

track obliterates within a few hours of removing the tube, in very chronic cases it remains patent and can only be closed by excision of the walls. It follows that a surgeon who changes a tube during the early stages of treatment will be well advised to have a duplicate ready and sterilised so that it can be slipped in immediately. At the other extreme is a patient, operated upon eight years ago for multiple abscesses in the lower lobe of the lung and treated at that time by Graham's cautery lobectomy and drainage, who has a permanent sinus and has not used a tube for several years. This man remains at work in good health; a small amount of pus discharges freely from the sinus into dressings.

THE CARE OF TUBES USED FOR PLEURAL DRAINAGE.—It is not the purpose of this paper to discuss the relative merits of " intercostal," " closed " and " open " drainage. The statement that " closed " drainage can only be maintained for a short time, because infection causes necrosis around the tube and prevents it being air-tight, is not wholly true. If infection in the

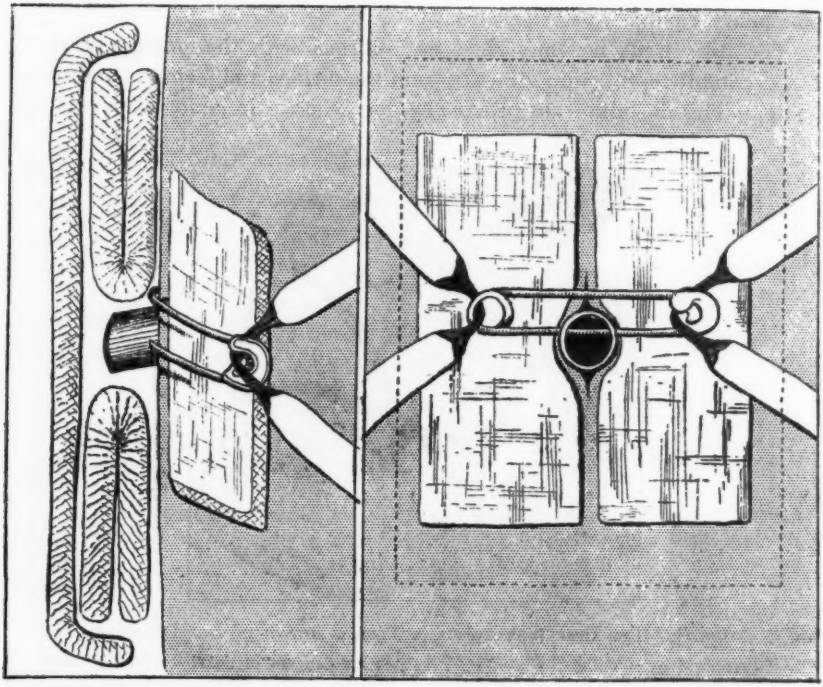

FIG. 4.—DIAGRAM TO ILLUSTRATE THE ARRANGEMENT OF GAUZE AROUND AN " OPEN " TUBE.

A small piece of gauze is placed on either side of the tube to protect the skin from the pressure of the safety-pin. Two more pieces of gauze are taken and folded upon themselves in such a way that, when laid above and below the tube, the superficial dressings are held clear of the opening. The object is to fix the tube securely without causing discomfort and without impeding drainage.

chest wall is mild, closed drainage of reasonable efficiency can be kept up as long as necessary, because there is a natural tendency for the tissues to close round the tube.

A great variety of drainage tubes have been devised, but the simple ones are the most practical and efficient. Choose a tube which is firm and whose lumen relative to the external diameter is large. Barium loaded

tubing is now available, and has the advantage of being opaque to X-rays.

When the tube is first introduced the end should protrude about 1½ inches into the pleural cavity and should not be in contact with any part of the wall of the empyema. It should be held in position by a safety-pin and strapping, and should not be sewn to the edges of the skin because the stitch is under tension and becomes septic. Unless the

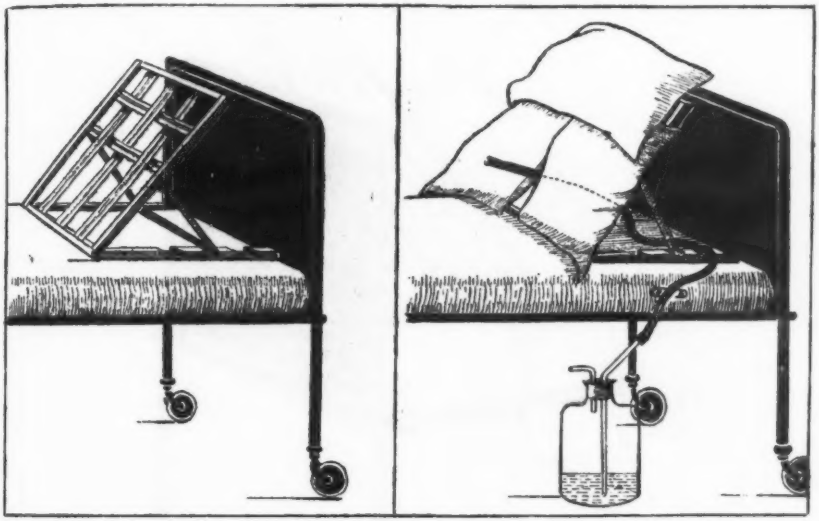

FIG. 5.— DIAGRAM SHOWING THE ARRANGEMENT OF BACK REST AND PILLOWS FOR "CLOSED DRAINAGE."

The tube passes directly backwards between the pillows and the canvas strips of the back rest. Note that the lowest horizontal strip of canvas has been cut out to allow the tube to move upwards or downwards as the patient changes position. Note also that ample slack should be available in the tube so that the patient can sit up. A tube which is deflected to one side, by pillows arranged in the usual way, is inevitably kinked and consequently obstructed.

tube is fixed securely there is a danger that it will be sucked into the pleural cavity and "lost."

The diagrams illustrate the nursing details appropriate to "open" and "closed" drainage systems.

In an article devoted to the care of patients with chest wounds Miss Gaffikin describes the obligations of the nurse in relation to the care of tubes. "The nurse should understand that perfect drainage is the most important part of the patient's treatment, and that after the tube has been correctly placed by the surgeon the responsibility for this is largely hers.

Continuous free drainage may make a difference of weeks or months to the time the patient spends in hospital. Such points as nursing the patient in the position most efficient for drainage, and seeing that the tube never

FIG. 6.

A patient treated by "closed" drainage should be comfortably seated in bed, and should sleep in this position. The same is true in the case of "open" drainage. This is the only way of maintaining continuous dependent drainage. The tube should not hang in a loop between the safety-pin, which fixes it to the bed, and the empyema bottle.

becomes kinked or pulled out when the patient moves and makes it taut, should be borne in mind. . . . It is always easy to tell if the tube is draining freely, because with each respiration the fluid in the empyema bottle is drawn

a little way up the glass tube on inspiration and falls again on expiration. Nurses should be instructed to observe this as they go about the ward, and if they acquire the habit of glancing at the bottle each time they pass the patient it is unlikely that the tube could be blocked for long. . . . The amount of drainage should be measured every day and a record kept on the chart. . . . The connecting tube and the empyema bottle should be

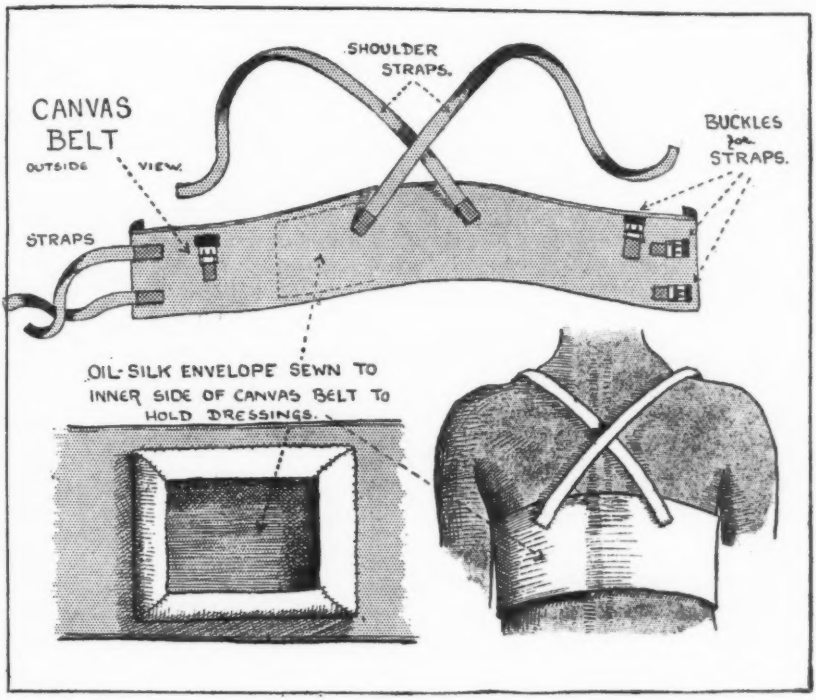

FIG. 7.

In the early stages of treatment the dressings are best secured by a many-tail bandage. The belt in the diagram is a simple and convenient way of fixing dressings in patients who have a small amount of discharge and are at work. The advantage of such a belt is that the dressings are not likely to shift with active movements.

cleaned every day by disconnecting them at the point nearest the chest wall after the tube from the pleural cavity has been clipped off."

Active movements should be encouraged throughout the whole period of " closed " drainage; apparatus which anchors the patient to a fixed position is a bar to progress and most tedious to endure.

A tube in the pleural cavity is no contra-indication to intensive inspiratory breathing exercises, and the patient should get up as soon as possible

after " open " drainage has been instituted. In many cases heavy manual labour can be profitably undertaken during the final stages of drainage.

ABNORMAL CONDITIONS OF THE CHEST WALL ASSOCIATED WITH TUBES.—*Inflammation of the chest wall* is of various types.

When an acute empyema is drained the tissues of the chest wall are contaminated by organisms from the pleural cavity and some measure of suppuration is inevitable. It can be minimised by attention to detail at the time of the operation. A limited incision should be used, and the initial pleural opening should be small; the nozzle of a suction tube introduced into the pleural cavity through this hole prevents the pus flowing out over the wound. The wound should be closed with the minimum number of stitches, and not sewn tightly round the tube.

Osteomyelitis of the rib is another possibility, and small sequestra of bone are often shed into the pleural cavity during convalescence; these act as foreign bodies and are a cause of chronic empyema. Some surgeons attempt to avoid this complication by coating the cut ends of the rib with Horsley's wax before the pleura is incised.

Cellulitis or erysipelas can occur at any time during convalescence and is generally due to inefficient drainage. The usual reason is that the tube is blocked or the end projecting above a collection of fluid in the cavity. ***Whenever pus escapes around the tube, instead of through it, it is safe to assume that something is wrong with the drainage.*** Cellulitis may start in the subcutaneous tissues or in the deep part of the tube-track, and in the latter event the infection spreads in the loose areolar tissue between the large muscles of the back. The following case illustrates this point. A patient was admitted with a diagnosis of chronic empyema and uræmia; the latter was suggested because there was generalised œdema of the back extending to the buttocks, albuminuria, drowsiness, and a dry tongue. The " œdema " was in fact due to streptococcal pus which had tracked downwards beneath the latissimus dorsi and become superficial by passing through Petit's triangle; the constitutional signs of uræmia were part of the toxæmia of streptococcal cellulitis. The patient was successfully treated by multiple incisions to drain the pus, sulphanilamide therapy, and correction of pleural drainage.

Spreading gangrene of the skin is a rare but serious complication of drainage. It is thought to be due to a symbiosis of a specific hæmolytic micro-streptococcus and a non-specific staphylococcus, although many other organisms have been described. It affects the skin only, and generally occurs, in old or debilitated men, a few weeks after drainage. The earliest signs are redness and swelling around the tube, and from this beginning a carbuncular lesion forms and spreads concentrically at the rate of a few

PLATE XIII

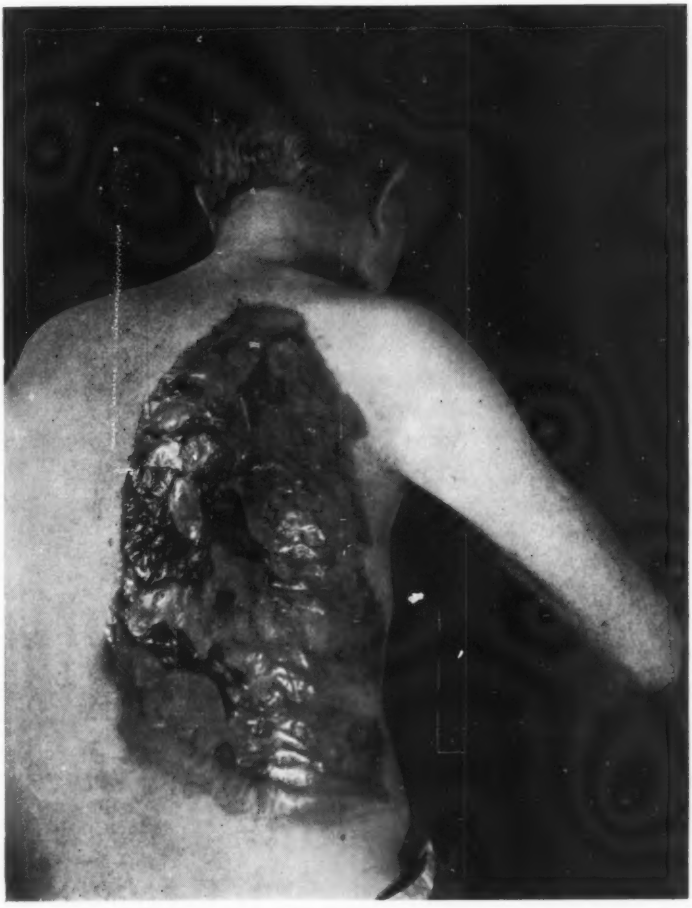

FIG. 8.—PHOTOGRAPH OF A PATIENT WITH SPREADING GANGRENE OF THE SKIN WHICH STARTED IN A STITCH HOLE NINE DAYS AFTER DRAINAGE OF A STREPTOCOCCAL EMPYEMA.

The patient was eventually cured by excision of the gangrenous area.

(J. W. S. H. Lindahl, St. Thomas's Hospital Reports; 2nd series, 1936, vol. i., p. 185).

[*To face page 70.*

millimetres a day. The advancing edge is raised and menacing; it leaves discoloured, dead and leathery skin in its wake, and under this a thin stratum of pus which seeps out spasmodically. Red granulations cover the subcutaneous tissues beneath the slough. The general condition of the patient is not affected at first and there is no toxæmia, but pain is constant and progresses until the patient becomes exhausted and dies. The majority of fatalities occur more than six months after the necrosis starts. Local applications, with the possible exception of zinc peroxide (Meleney), are powerless to check the gangrene, and only two methods of treatment are likely to be successful. The first is the scientific use of maggot therapy, and the second is to remove the tube and excise the necrotic area by cutting through normal tissues beyond and beneath it. The patient should be given adequate doses of sulphanilamide, and the raw area, left by removal of the slough, can often be successfully covered with a skin graft.

Painful Tubes.—When a patient complains that a tube is painful, the inference should be that adjustments are needed. It is all too common to be asked to see a patient whose tube is painful and whose progress is unsatisfactory, only to find that the tube has come out of the pleural cavity and is merely sticking into the chest wall.

Other common causes of pain are that the strapping which fixes the safety-pin in the tube has been applied with uneven tension and is dragging it towards one side of the wound. Exuberant granulations are evidence of mild local sepsis, and pain in these cases is due to pressure upon the inflamed area. Healthy granulations have a flat, red surface and are painless.

The sinus sometimes contracts and presses upon the tube at a time when drainage is still essential and when a smaller tube cannot be substituted with safety. This causes pain which should be relieved by introducing a laminaria tent to dilate the sinus and so enable the tube to be reinserted comfortably.

The above sources of pain can easily be corrected, but there are some patients who, in spite of all attention, experience intractable discomfort, and in these the treatment is to inject the relevant intercostal nerve with alcohol. The nerve is most accessible at the angle of the rib beneath which it passes to the subcostal groove. The angle of the rib is about 2½ inches from the mid-line of the back. Using local anæsthesia and a hypodermic needle, raise a weal in the skin; push inwards until the needle comes against the rib and inject 1 c.c. of local anæsthetic into the intercostal muscles below the rib. If pain is relieved within a few minutes, inject 1 c.c. of 90 per cent. alcohol into the same place. Alcohol should never be injected without preliminary novocaine anæsthesia because it produces

intolerable pain; if exception be taken to the use of local anæsthesia because it dilutes, and so prejudices, the effects of alcohol, the injection of the latter should be done under a general anæsthetic.

ABNORMAL INTRATHORACIC CONDITIONS PRODUCED BY TUBES.—The majority of these conditions are due to pressure of the tube upon important structures. The diaphragm is apt to rise and come into contact with a tube which has been inserted too low in the chest. It may block the orifice of the tube or cause pain in the tip of the shoulder on the same side. This pain is often diagnosed as " rheumatism "; it is due to stimuli transmitted to the fourth cervical segment by the phrenic nerve and referred to the area of skin supplied by the lateral supraclavicular nerve. The treatment is to adjust the tube and possibly to redrain the pleura at a higher level.

As the empyema resolves the surface of the expanding lung is apt to come into contact with the drainage tube. If the lung presses upon the side of the tube no harm accrues, but if the tube is rigid and its end butts against the lung a broncho-pleural fistula or pulmonary suppuration is inevitable. A case was admitted recently in which the tube had ulcerated into the lower lobe bronchus, and remedial treatment was difficult and prolonged. These accidents can be avoided by using a tube which is slightly curved and which inclines upwards in the pleural cavity parallel to the chest wall.

Broncho-pleural fistula is generally not due to ulceration of the tube into the lung; it is a legacy of the original pulmonary lesion which caused the empyema. Small communications are probably common and pass unnoticed because they are symptomless; fistulæ of moderate size can be recognised because air gushes out of the tube when the patient coughs or raises the intrabronchial pressure by closing the nose and mouth and attempting " to blow "; large fistulæ are obvious because air passes in and out of the tube every time the patient breathes. A large fistula is very inconvenient to a patient with pleural drainage because cough is ineffective and dyspnœa accompanies every exertion. Special tubes containing valves, which permit pus to escape but prevent air entering the chest, have been devised, and the simple expedient of gluing a perforated finger-stall to the chest wall round the sinus has been recommended. These procedures are unsatisfactory and impede free drainage.

The following case is a catastrophe of another type. A young girl was operated upon for total unilateral bronchiectasis of the left lung, and pneumonectomy was successfully performed. At the end of the operation a tube was inserted into the lower part of the pleural space and " closed " drainage established. Convalescence was uneventful for five weeks and the general condition of the patient was excellent; " open " drainage had been

substituted for the original tube and the space from which the lung had been removed was already much reduced by movement of the mediastinum and heart towards the affected side. At the sixth week there was a sudden discharge of clear fluid from the wound and it was assumed, at that time, that a sterile loculus in the pleura had ruptured into the main drainage space. The patient, however, became gravely ill and died, and the explanation was not discovered in spite of repeated clinical and X-ray examinations. At the post-mortem the tube was found to have ulcerated into the pericardium and caused a serous effusion which had ruptured and become infected with organisms from the empyema.

A rare but important lesion is diphtheritic infection of the wound and empyema cavity. It generally occurs as the result of an outbreak of the disease in a ward, but can occur in sporadic cases. In a recent epidemic two types of infection were observed. In the one there was a definite membrane in the wound and the patients had the constitutional signs of diphtheria; in the other the wounds looked healthy, there were no general signs, but pathological organisms were present. The gravis strain of organism was not confined to the cases with membranes, nor was the mitis strain peculiar to wounds of healthy appearance. All patients in the ward, as well as medical personnel and nurses, should have throats, noses and wounds examined by the wet-swab technique; positive cases should be isolated and treated with the appropriate serum and sulphapyridine snuff applied to the wound. The condition is apt to prove fatal in cases with a definite membrane. The frequency of diphtheroid organisms in the lung and pleural cavity makes it essential to establish the virulence of the organisms by laboratory tests.

The Signs and Symptoms of Inefficient Drainage.—Efficient drainage is characterised by rapid improvement in the local and general conditions. The converse is also true, with the proviso that changes for the worse are of insidious onset and apt to pass unnoticed at first. The earliest symptoms are generally reported by the nurse, who finds that the patient is difficult to make comfortable in bed, loses appetite and feels off colour. Tachycardia is an early and important sign. The quantity and quality of pus discharged from the tube may change and the patient start to cough and bring up sputum. The combination of tachycardia, pyrexia, increase of cough and sputum associated with diminution or alteration of pus discharged from the tube is pathognomonic of inefficient drainage. The first hint of unsatisfactory drainage should lead to clinical and X-ray examinations being undertaken.

Causes of Inefficient Drainage and their Correction.—*Blockage of the tube* is generally due to the fact that it is linked by faulty position of

the patient as he lies in bed. Pus or debris from the cavity impacted in the tube can be cleared by milking a " closed " drainage tube towards the empyema bottle, or by irrigating and gently passing a bougie along an " open " tube. If these simple procedures are ineffective, the tube is blocked either by granulation tissue which has grown into a hole cut in its side or because its end is in contact with the diaphragm or cavity wall. In both cases the tube should be changed and its position adjusted. *It is a good rule never to cut a side hole in a tube set to drain a small pleural pocket*; granulation tissue grows into side holes.

A tube whose lumen is small is particularly liable to get obstructed, and for this reason intercostal drainage becomes unsatisfactory as soon as the pus thickens. The space between ribs is inadequate to allow the passage of a large tube, and few cases of empyema resolve without rib resection at some stage.

The tube is not well placed for drainage. This point can only be verified by postero-anterior and lateral pleurograms. In the period immediately after operation difficulties do not arise on this account, but as the cavity shrinks in size the margin of error is likewise reduced.

The correct size, length and position of a tube are as follows: The size depends upon two factors. The first is the thickness and viscosity of the pus to be drained, and the surgeon should satisfy himself that the lumen is adequate to transmit discharges freely from the cavity without repeated obstructions. The other factor is equally important but more controversial. There is evidence to suggest that in the early stages of drainage, be it open or closed, the internal diameter of the tube should not be less than $\frac{1}{2}$ inch. A smaller tube prevents air escaping from the pleural cavity quickly enough to avoid some measure of positive intracavitary pressure at the end of expiration, and so puts a brake on pulmonary expansion. The writer suggests that this accounts for the fact that the mere introduction of a slightly larger tube into an empyema which seems to be well drained, but in which resolution is unduly slow, may make all the difference to subsequent progress. It also may bear upon the inability of an intercostal tube to cure the majority of empyemata without subsequent rib resection. The point is most relevant in the early stages and becomes less important as time passes. A tube of excessive size is harmful because it promotes and aggravates sepsis in the chest wall and the empyema cavity. The tube should be long enough to traverse the whole length of the sinus through the chest wall, and its tip should lie at the bottom of the pocket to be drained. If it is too long, pus either accumulates and discharges by overflow or seeps into the subcutaneous tissues around it. These details are commonly ignored in practice and account for many cases of chronic empyema.

Total empyemata are generally obliterated from above downwards because the apex of the lung expands before the base, and consequently the last pocket is conveniently placed for drainage in the lower and posterior part of the pleural cavity. If, however, the base of the lung expands before the apex the main cavity draws away from the tube, becomes separated and sealed off. This complicates drainage and is unfortunately common in traumatic infected hæmothorax; it is explained by the fact that partial atelectasis of the lung accompanies a high proportion of the cases and re-expansion is unduly slow. There are two ways of overcoming the difficulty.

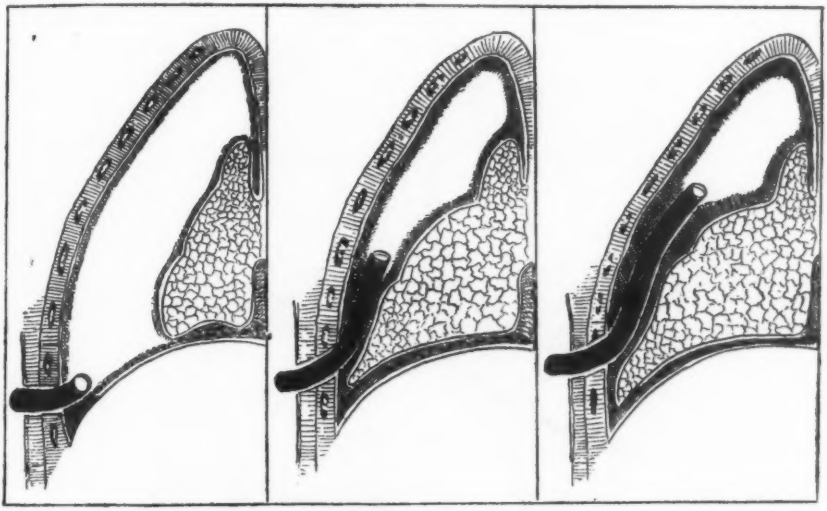

FIG. 10.—DIAGRAM TO ILLUSTRATE AN EMPYEMA WHICH IS OBLITERATING FROM BELOW UPWARDS.

The best way to maintain efficient drainage is to follow the cavity up by repeatedly lengthening the tube. When the main cavity is finally closed the tube can be rapidly shortened and removed; its track heals without difficulty.

The first is to increase the length of the tube repeatedly so that it follows the retreating cavity upwards, and in these circumstances the track through which it passes is merely a granulating sinus which closes as soon as drainage is concluded. The other alternative is to redrain the cavity high up in the axilla or behind the vertebral border of the scapula, and neither of these operations is to be recommended.

Empyema cavities tend to become bilocular and the tube to drain one part to the exclusion of the other. Lateral pleurograms often show a track passing from behind forwards over the surface of the diaphragm and

another branching upwards towards a cavity in the paravertebral gutter. The tube tends to find its way into the horizontal limb, and the upper pocket either drains poorly or becomes sealed off. It is important to manipulate a tube around the corner into the upper cavity, and if the other part is of any size it should be drained by a separate incision at the front. There is a tendency for any empyema to loculate and to form pockets at some distance from each other; these may be infected by different organisms.

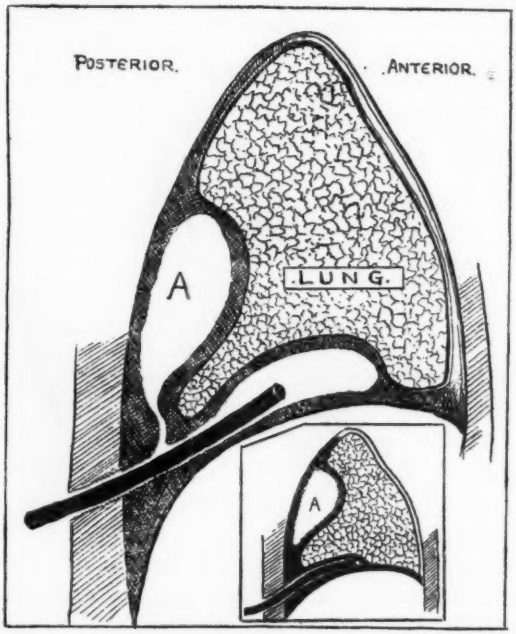

FIG. 11.—DIAGRAM SHOWING A BILOCULAR EMPYEMA OF A COMMON TYPE.

Unless the presence of the upper pocket lying in the paravertebral gutter (A) is ascertained by pleurograms, the communicating track will close off and a closed chronic empyema will result. A tube must be "juggled" round the corner into the upper pocket.

The diagnosis of loculation is suggested by the general signs and symptoms of inefficient drainage occurring in a patient whose main cavity is known to be well controlled; it is confirmed by X-rays and aspiration of pus from the suspected area. A second drainage tube should be inserted.

If an empyema persists for a long time the walls become very "thick," because layer upon layer of fibrin is deposited and organises. It follows that a tube which just protrudes into the cavity at the beginning of drainage is likely to get left behind in the chest wall as time passes, and the policy of

shortening the tube as the discharge diminishes is unsound. Chronicity also leads to fibrosis in the endo-thoracic fascia, and this approximates the ribs. The ribs themselves are affected in time; they become roughened and triangular in cross-section, and ultimately fuse together to form a solid carapace of bone. These advanced changes interfere with drainage and presage further operations to close the cavity.

ANCILLARY TREATMENTS.—*Irrigations of the cavity* are sometimes used to supplement drainage. Many solutions have been advocated from time

FIG. 12.—FOUR RIBS REMOVED FROM THE CHEST WALL OF A PATIENT WITH A CHRONIC EMPYEMA.

Note the triangular shape of these ribs and the fact that they are fused together. These changes produce a rigid hemithorax and generally prevent the healing of an empyema.

to time, but those in common use are saline, Dakin's solution, Milton, and azochloramide. The technique of irrigation is simple; the patient is laid in such a position that the drainage tube is uppermost and fluid is run into the pleural cavity. No attempt should be made to fill the space to overflowing, and when a reasonable amount has been introduced the patient is turned so that the tube is dependent and the fluid runs out. Some methods of doing this with tubes of various types are illustrated in the accompanying diagrams.

There are two fundamental points which must be considered before irrigation is used. The presence of a broncho-pleural fistula is an absolute contra-indication. To avoid the risk of fluid entering the bronchial tree through an undetected fistula a very small amount of fluid should be used on the first occasion. It should be non-irritating to the bronchial mucosa and should have a taste. The first indications of a fistula are that the

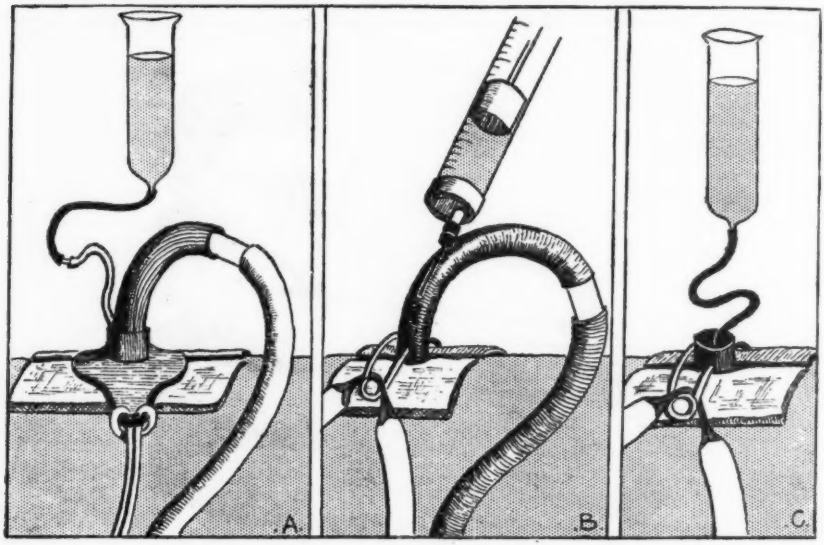

FIG. 13.—A. IRRIGATING AN EMPYEMA WHICH HAS BEEN DRAINED WITH A TUDOR EDWARDS TUBE.

Note the diameter of the irrigating tube is much less than that of the main tube; this obviates the risk of causing positive intra-pleural pressure in the cavity.

B. "CLOSED" DRAINAGE WITH A STRAIGHT SIMPLE TUBE.

The irrigating solution is injected into the main tube by inserting a needle into its lumen.

C. "OPEN" DRAINAGE.

Irrigation can be done by passing a fine catheter into the depths of the empyema cavity.

patient coughs and tastes the fluid. Irrigation should be stopped at once.

The second important point is that the intrapleural pressure should never be raised by irrigation; this necessitates having a patent and large exit, as well as the tube through which the fluid is introduced. The practice of connecting a syringe to the end of the main drainage tube is to be deprecated. The dangers of positive intrapleural pressure are air embolism, pulmonary injury, and disruption of adhesions limiting the inflammatory process.

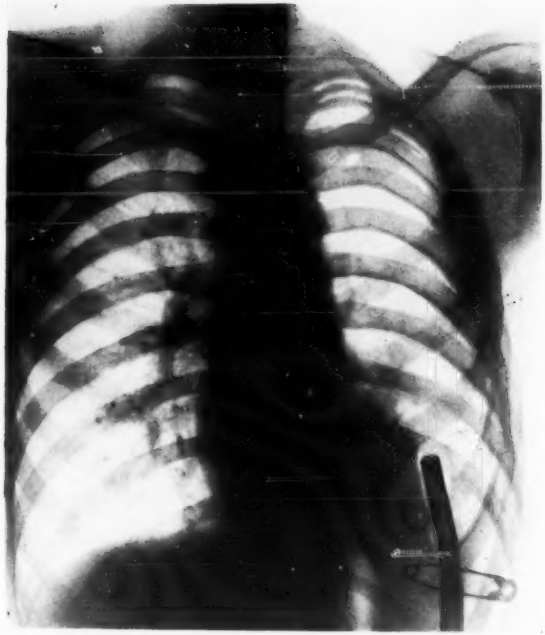

FIG. 15. In this X-ray it seems as though the empyema on the left side has resolved and the tube is only draining the small pocket at its tip. (The exposure on the two halves of the film is different.) If this were so, the time to remove the tube has come, but the point cannot be verified without a pleurogram. The tube shown in this X-ray is barium-loaded.

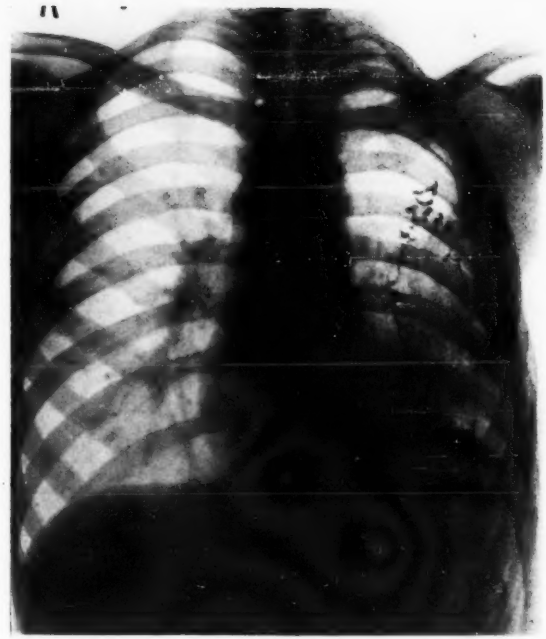

FIG. 16.—A PERFECT PLEUROGRAM OF THE SAME PATIENT, SHOWING THAT A CONSIDERABLE CAVITY REMAINS.

The ring marks the orifice of the sinus on the surface. Drainage must be continued. (Mr. C. Price Thomas's case.)

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Continuous suction is sometimes applied to chronic empyema cavities. This can only be done provided closed drainage can be kept reasonably air-tight, and a satisfactory suction motor is available. The most convenient apparatus is produced by the Genito Urinary Manufacturing Co., and is shown in the accompanying diagram. It has the merits of being silent, of requiring very little mechanical attention, and of keeping up a negative pressure of controllable degree for long periods of time. The disadvantage of continuous suction is that the patient is immobilised at a time when active

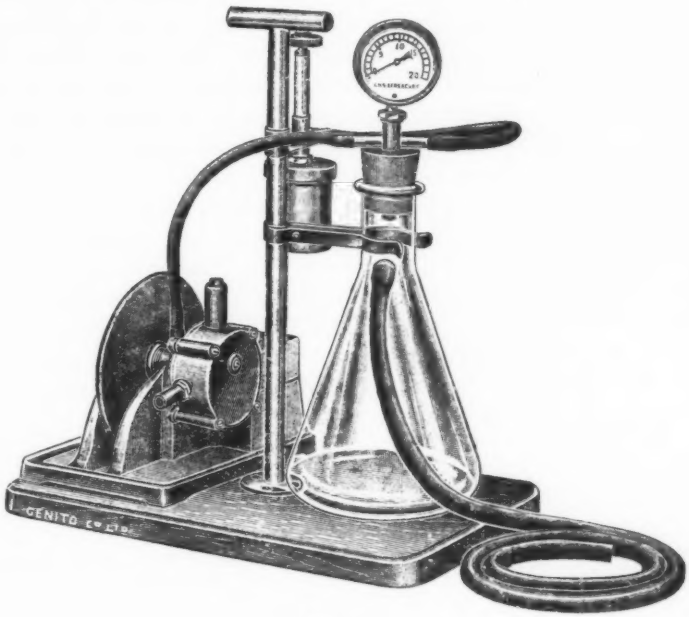

FIG. 14.

exercises are most needed; this disadvantage can be overcome to some extent by using Brock's portable apparatus.

FINAL REMOVAL OF THE TUBE.—The only way to determine the proper time to take the tube out is to make serial pleurograms during convalescence. These X-rays should demonstrate progressive diminution in the size of the cavity, and drainage is necessary until only the track of the tube remains. The volume of an empyema cavity decreases rapidly in the early stages of drainage, but final obliteration is often long and protracted.

Pleurograms are made by laying the patient on the normal side, taking out the drainage tube, and inserting a slender catheter into the depths of

the cavity. Lipiodol is injected through this catheter. If the oil is injected from a syringe into the drainage tube it may be prevented from reaching the main cavity by an air-lock. The object is to coat the walls of the space with the oil and not to fill it to overflowing. The catheter is then removed and a fine piece of leaded rubber equal in length to the drainage tube is fixed in the sinus; the orifice of the latter is plugged with gauze and a small metal ring is placed upon the skin to mark the orifice of the sinus. The patient is then turned about in various directions and X-rays taken in the postero-anterior and lateral positions. The films show the size and shape of the cavity and the leaded rubber demonstrates the lie of the drainage tube relative to the bottom of the empyema. The only danger of this investigation is the possibility that the patient may be sensitive to iodine. Both lipiodol and neo-hydriol contain 40 per cent. of iodine in poppy-seed oil. Reactions are fortunately rare, but occasionally cause serious constitutional signs such as pyrexia, anorexia, coryza, injection of the eyes, puffiness of the face, and dermatitis in the form of blisters, vesicles or pustules. Sensitivity to iodine can be tested in doubtful cases by giving potassium iodide gr. x. t.d.s. for twenty-four hours before doing a pleurogram.

Conclusion.—The difficulties and pitfalls of pleural drainage have been underlined, and in so doing the beneficial effects have been glossed over. In conclusion, therefore, tribute must be paid to the life-saving properties of a tube inserted at the time of election, watched and adjusted during convalescence, and removed at the moment of cure.

AN OUTBREAK OF INFLUENZA IN THE TROPICS

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In October, 1940, an outbreak of pyrexial illness occurred in one of His Majesty's ships anchored in harbour in the tropics, and a considerable number of the ship's company was affected. The disease was highly infectious and spread to two other ships. Cases also occurred among the sick-berth staff of the attendant hospital ship.

Facilities for nursing the sick on board the warship were very restricted and consequently all cases were transferred to the hospital ship within

PLATE XV

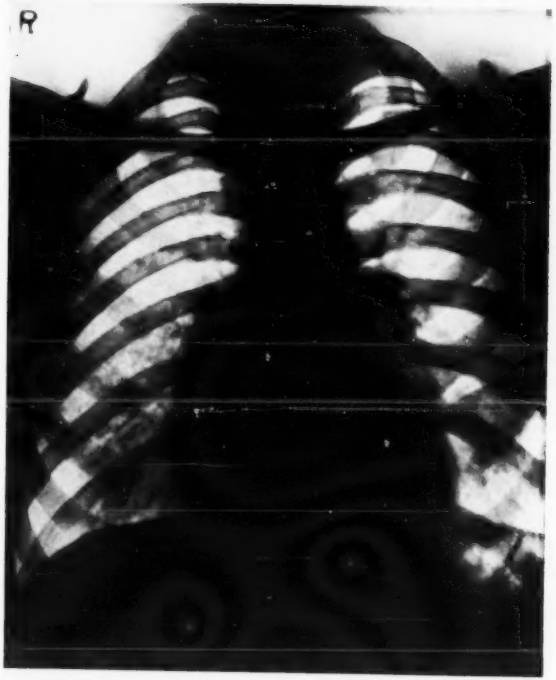

FIG. 2.

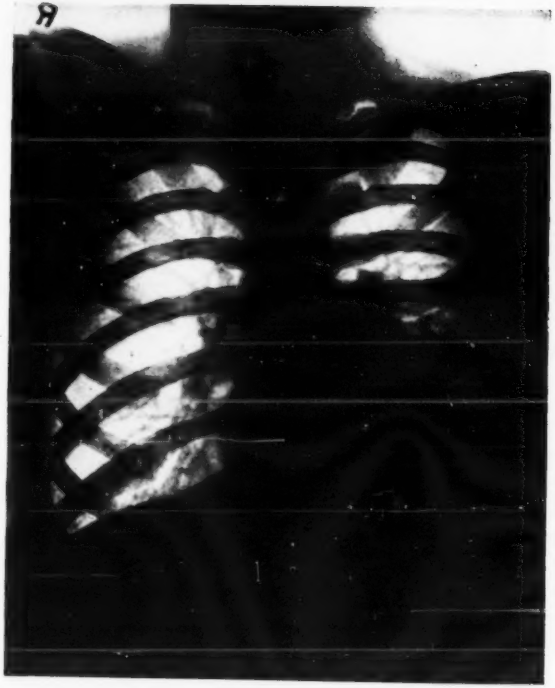

FIG. 3.

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twelve hours of the onset of symptoms. Also some men living ashore, where adequate treatment of sick persons was not available, were sent to hospital as soon as they reported sick. It will thus be be seen that a good opportunity presented itself for observing these cases from the beginning. A first-class X-ray department and bacteriological laboratory were incorporated in the hcspital ship and all patients were under observation until they had been apyrexial for some days and were fit for light duty.

A total of forty-seven cases were seen, and they have been divided into four groups: (1) mild cases, numbering thirty, (2) moderately severe cases with abnormal pulmonary signs and symptoms, numbering nine, (3) severe cases with pulmonary complications, numbering seven, and (4) one severe, uncomplicated case. All these cases occurred within six weeks, between mid-October and the end of November, and almost all of them came from the same three ships, with the exception of one nursing sister, six sick-berth attendants and two members of the crew of the hospital ship.

The onset was similar in all cases and was very acute; thus many of the men went on morning parade quite fit but had to fall out later. The first symptom was usually a rigor, followed quickly by intense headache, giddiness and weakness. Initial temperatures were commonly about 102° F., rising rapidly within the next few hours to 104° F. On arrival in hospital ship, usually within twelve hours of the onset, the patient was very miserable, but not alarmingly ill. Headache was intense, frontal in localisation and often described by the patient as " behind the eyes "; actual eye movements were extremely painful. Vomiting was not a feature of the illness. About this time general limb pains became pronounced; the legs were affected more severely and more commonly than the arms, the pain being largely muscular in origin. Lumbosacral backache was a very constant feature. No swelling or limitation in movement of the joints was noticed.

Rigors were uncommon after the first twelve hours, so much so that if they occurred the diagnosis was probably a wrong one and further blood smears were examined for malarial parasites, even though the initial tests had been negative. Since most of the cases were sent in to the hospital ship with a clinical diagnosis of malaria—a perfectly justifiable one under the circumstances—at least three blood smears were examined at intervals, and often many more, before malaria was ruled out. The work was incidentally made much simpler for the medical officers concerned because no prophylactic quinine was at that time being given to these men.

Physical examination at this, the early stage, revealed a grossly furred but moist tongue, and very little else abnormal. A few cases only complained of sore throat, thus contrasting with reports of epidemics of the last few years, in which tonsillitis and pharyngitis were prominent. Nor was

there any evidence of nasopharyngeal catarrh, sinus infection or middle-ear disease. The patient was more co-operative than his malarial counterpart, and less sleepy; there was not the heavy, injected, blurred look about the eyes, which in conjunction with a suffused face is so typical of malaria. The optic discs were normal; the heart and lungs were normal, and no enlargement of the spleen was noticed. Although the temperature might be 104° F., or higher, the pulse rate rarely rose above 90.

After twenty-four hours, in almost every case, including the mildest, the patient complained of a "sore feeling" in the substernal region, together with an irritative cough, at first unproductive, but after a further twelve hours usually giving rise to a sticky, mucopurulent sputum. Examination of the lungs at this stage might show occasional scattered rhonchi, but more often than not no abnormal signs could be detected. The progress of the illness is best shown by the accompanying temperature chart (Fig. 1), which is typical of the mild case complicated only by a short-lived tracheitis and bronchitis. As will be seen, the temperature returned to normal on the seventh day and the patient was able to get up twenty-four hours later and return to light duty after a further three days.

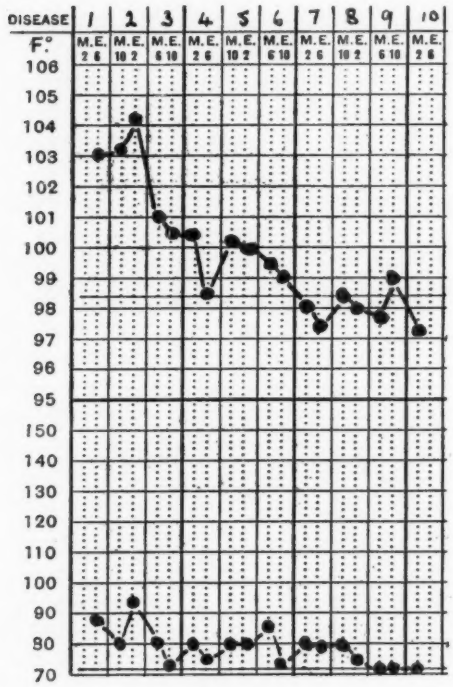

FIG. 1.

Treatment of these mild cases was symptomatic. A.P.C. powders were found satisfactory for the headache and a simple linctus relieved the irritative cough for two hours or so, but, as will be seen in the severer cases, the cough was extremely resistant to treatment. The last symptom to disappear was usually the headache, which was often present to a troublesome degree after the temperature had fallen, especially early in the morning for a few hours. No nervous signs or symptoms were observed and there was no post-influenzal depression, tachycardia or bradycardia.

The great majority of the cases were similar to those described above. In nine, however, pulmonary signs and symptoms became the main feature of the illness. The onset was the same—*i.e.*, an acute constitutional malaise, followed by a tracheitis as before; but this rapidly developed into a pneumonitis which was almost always localised to the base of the left lung. At no time was the patient really ill, but a productive cough was always the predominant symptom after the first twenty-four hours, and proved very distressing. There was no respiratory distress of any kind. The pulmonary signs at the affected base consisted of an impaired percussion note with diminished breath sounds and coarse, moist crepitations. The pyrexia lasted only a short time, about one week, but, in contrast to the cases in the first group, cough would persist for a further week, together with the physical signs. The cough was practically confined to the night-time and hourly bouts would recur until daylight. Sputum would be mucopurulent in type, very viscid and very profuse, the volume being greatly increased by large quantities of "froth." No hæmoptysis was observed at any time. X-ray examination of the chest showed bronchopneumonic mottling at the affected base of the lung (see Fig. 2, Plate XV). These cases responded well to an expectorant mixture given during the day and to a strong dose of linctus heroin at night, and they were all fit for light duty after about two weeks.

In the third group we have seven cases which were much more severe, but which gave rise not so much to prognostic anxiety as to therapeutic difficulty. The initial stages were identical with those previously described, but respiratory symptoms and signs were much more pronounced. Both lung bases were usually affected, the temperature took longer to settle and the physical signs and cough were very persistent.

Of these seven cases, three were a source of considerable worry and will be described in more detail, together with the bacteriological and radiological findings, as they present contrasting clinical pictures.

The first, A. T., aged 18, started his illness typically, but his signs were bilateral, affecting the lower half of each lung. They were patchy and of a bronchopneumonic rather than a lobar pneumonic type; there was no pleural pain, respirations were only slightly increased, and cyanosis, though present, was not marked. The alæ nasæ were working. X-ray showed a bronchopneumonic mottling affecting the left lower and mid-zones; the right lung was radiologically normal, although physical signs were marked. No sputum was produced at any time, so that lung puncture was resorted to in order to identify the organism. The alveolar fluid obtained by the exploratory needle gave on culture a pure and heavy growth of staphylo-

coccus. The patient made a good recovery on symptomatic treatment after an illness lasting three weeks.

The second patient, R. S., aged 19, developed typical symptoms of influenza, which was soon complicated by respiratory infection. In this case the signs were very localised, and were all at the left base. The general picture was one of a moderately severe lobar pneumonia: the patient's facies was grey and anxious; respirations were distressed and accompanied by severe pleural pain; the cough was troublesome and sputum plentiful, but not bloodstained. When cultured on blood agar it gave a growth of staphylococcus and *H. influenzæ*. The latter grew in typical, translucent, satellite colonies round the staphylococcal growth. In view of the toxæmia and in spite of the unpromising bacteriological findings it was decided to try the effect of sulphapyridine, with inconclusive results. The patient returned to light duty after five weeks. An X-ray taken before treatment was started (Fig. 3, Plate XV) showed an inflammatory opacity at the left base, suggesting a lobar consolidation rather than a bronchopneumonia.

The third case, V. B., aged 22, had suffered a moderately severe influenzal bronchitis with signs at both lung bases, from which he was making a good recovery and was about to be discharged, when he developed signs of a pleural effusion on the right side. Exploratory puncture revealed a moderately clear fluid which was sterile on culture but which contained polymorphonuclear cells. The fluid remained sterile for two weeks but was greener in colour and more opaque than the ordinary serous effusion. It finally became frankly purulent, the pus giving a pure culture of staphylococci. A satisfactory recovery was made after surgical drainage.

The remaining four cases in this group showed nothing exceptional beyond bronchopneumonic changes in the lungs, but they set a problem in therapeutics by reason of their persistent cough. This was confined almost without exception to the hours of darkness and was accompanied by the usual sputum in considerable quantities. The ordinary heroin in large doses was effective for about two hours, but three or four doses might be required during a restless night. Morphia was singularly ineffective besides being inadvisable; inhalations of benzoin and menthol, in combination or singly, seemed only to aggravate the condition. Quinine, although it produced good results as regards the general condition and temperature, seemed to have no effect on the cough itself. Finally, in desperation, five of the worst "coughers" were removed to a small ward which was originally meant for tuberculous patients and which was the coolest and airiest in the ship. This move resulted in immediate symptomatic improvement without further treatment, and it is felt that these patients might be nursed as far as possible day and night in the open air.

The fourth clinical group differs from the remainder in that severe constitutional symptoms were prolonged without the intervention of any complicating conditions.

J. W., aged 25, was taken ill suddenly with headache and attacks of shivering thirty-six hours before admission to hospital ship. He had not been taking any prophylactic quinine. Examination on the second day revealed no abnormal physical signs other than an injected throat. His temperature was 102·8° F. and pulse rate 100. There was a slight, dry cough, but the only complaint was of headache, which was frontal and very intense, any movement of the head being resented. Repeated blood examinations for malarial parasites were all negative. The temperature, taken four-hourly, showed no deviation from the 103° F. mark for the next four days, during which time the patient's condition remained unchanged although it did not give rise to any anxiety. X-ray of the lungs showed nothing abnormal: total white cell count was 14,800 per c.mm., 82 per cent. of which were polymorphs. After taking blood for culture it was decided to try the effect of sulphapyridine, and the patient was given four-hourly doses of 1 gramme by mouth. On the second day of drug treatment his condition improved and the temperature began to fall by lysis (Fig. 4). This improvement was maintained, for, although he had to be returned to his ship at short notice, it was learned later that he made an uninterrupted recovery without any complications. The blood taken for culture showed a pure growth of *H. influenzæ*, the identity of the organism being confirmed by expert opinion ashore.

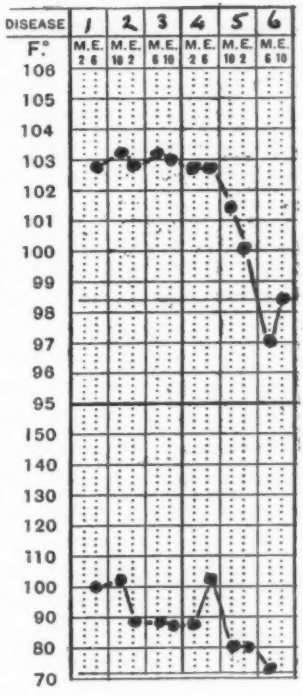

FIG. 4.

Routine urine examinations were done in every case, including centrifuging and microscopy of the deposit, but nothing abnormal was found in any of them.

Clinical Discussion.—From the clinical description given above, one or two points are worthy of stress. First, the periodic cough was always much worse at night and often quite absent during the day. This did not seem to be due to posture, as many of the patients tried every position without

relief. Open air was the most effective therapeutic agent. Secondly, the physical signs were extraordinarily constant, and varied only in the extent of lung involved. In patients with unilateral disease, the signs were left-sided in every case except one. The percussion note over the affected area was impaired, vocal resonance was diminished, breath sounds were weak and râles were coarse and moist. In only one case, that of R. S., did the signs resemble those of a pure consolidation. These findings correspond very closely to those of Chickering and Park,[1] who described just such an "atypical type of pneumonic involvement." Scadding,[2] in his treatise on the lung changes of influenza, suggests that these signs represent "an œdematous sodden condition of the lung bases, short of actual consolidation." In other words, a pathological state somewhere between a bronchitis and a true pneumonia. He also draws attention to the fact that the death-rate is closely related to the number of cases showing actual consolidation, a mortality of 37 per cent. amongst such cases being present during the epidemic of 1936-37. Such high figures are also quoted by French[3] and others for the 1918 pandemic. This tropical outbreak follows the rule with only one case of consolidation and no mortality.

It is, perhaps, pertinent at this stage to compare the clinical picture of the respiratory complications seen in this outbreak with those recently described by Hubble and Osborn[4] as occurring in children and infants. These observers draw attention to the probability of an acute bronchiolitis being the predominant lesion in influenzal infections, especially in infants, and also probably in adults. The main symptom of this bronchiolitis is gross respiratory distress with marked cyanosis, producing a picture of "obstructive dyspnœa." They also state that the percussion note was usually more resonant than normal owing to the emphysema secondary to local areas of collapse. Obviously such a clinical lesion is vastly different from the one described above, but there are certain points of resemblance. Thus the early tracheitis, followed quickly by a profuse secretion, is common to both types, only in adults the danger of "self-drownage" is very much less than in infants. The profuse expectoration which was so noticeable in even the mildest of the adult cases in this series, with minimum signs and no respiratory distress, may possibly be explained by a localised area of bronchiolitis, although in fact it probably came from the trachea. But in the severer cases with extensive signs respiratory distress was not nearly so marked as Hubble and Osborn ascribe to bronchiolitis. Perhaps the forcible expectoration of the sputum by the adult is the determining factor in relieving distress, compared with the poor and easily tired "cough effort" of the infant.

Impaired percussion note, in some cases involving large areas of one or

both lungs, suggests either alveolar exudate or alveolar collapse. If the latter, then the bronchiole obstruction should lead to considerable distress, aggravated further by secondary emphysema, as is well illustrated in the cases of Hubble and Osborn. This distress was not present, and it seems that in the severer cases at least the alveolar exudate represented Scadding's œdematous state, the absence of any marked dyspnœa being due to the very low-grade toxicity of the infection. No mention is made by Hubble and Osborn of the bacteriology of their cases and it seems that on this point the two types would demonstrate an important contrast.

Finally, it is interesting to note that depression was conspicuous by its absence in this epidemic. Many authorities quote the resulting depression as one of the hall-marks of a true influenzal infection, but in no case did it develop in spite of war conditions on a station which was climatically uncomfortable and which was generally unpopular amongst the serving personnel.

Differential Diagnosis and Treatment.—The differential diagnosis of influenza in a tropical country brings up many interesting points.

First and foremost it has to be differentiated from malaria. On this particular station in October and November subtertian malaria is widespread. Clinically one could not be sure of the diagnosis, but the malarial patients were usually more miserable, drowsier and less co-operative, and vomiting was frequently severe and persistent for the first two or three days. This was uncommon in the influenzal cases. The latter did not, however, have the periods of symptomatic improvement which the malarial patient shows when his temperature falls, only to rise again subsequently; rigors were uncommon after the first twenty-four hours. No splenic enlargement was observed in any of the influenzal cases. In the first attack of malaria the spleen is sometimes enlarged, but by no means always. The only certain method of distinguishing between the two conditions was, of course, a blood examination. Thick films stained with Giemsa solution were used throughout and negative cases were repeated at least three times. It is interesting to recall that in a case of subtertian malaria in the acute stage, with a high temperature and without any treatment, the parasites at times disappear entirely from the bloodstream. It happened time and again that an obvious case of malaria, with severe pyrexia, admitted to hospital ship in the afternoon showed no parasites in the blood. When the examination was repeated a few hours later they would be present in very large numbers. The work of blood examination was made easier since none of the naval ratings was receiving prophylactic quinine.

The other condition, common on this station, which closely resembled influenza, was acute pyelitis. Contrary to experience in the United Kingdom,

urinary infections in the young adult male were frequently seen. These, curiously enough, gave rise to few urinary symptoms—no frequency and no dysuria—but constitutional disturbances included high fever, rigors, vomiting and backache. The differential diagnosis depended entirely on microscopic examination of the urine and, if necessary, midstream culture. With regard to the interpretation of pyuria on this station, it is interesting to note that a certain number of pus cells in a centrifuged deposit would frequently be found in various non-urinary diseases and also in normal individuals. This led the civil authorities ashore, some years previously, to investigate the question of urinary infection, and many normal healthy Europeans were found to have low-grade infections, chiefly staphylococcal.

Acute lobar pneumonia in the early stages is another condition easily confused with influenza, but only one case was seen during fifteen months. This is remarkable since pneumococcal infections are common amongst the native population.

Meningococcal meningitis may also simulate influenza very closely, and one such case was seen where meningeal signs did not make their appearance till some hours after admission. Lumbar puncture clinched the diagnosis. Miliary tuberculosis may also be mentioned here as a possible cause of diagnostic error.

Among the tropical diseases, other than malaria, from which influenza has to be differentiated are typhoid, typhus, dengue and yellow fever. Of these, typhus and dengue were absent from the district, anyhow during that particular year, and no case of typhoid was seen among the Service personnel. Yellow fever was present among the natives of neighbouring territories, but there had not been a case in a local European for some years past.

In more temperate climates influenza would have to be distinguished from such conditions as sandfly fever, tick fever, undulant fever and trichiniasis. The first three of these were unknown in this particular locality, but the last one might presumably be encountered. If so, a blood count should settle the diagnosis.

In most of these tropical diseases, apart from exceptional cases, the diagnosis should not be difficult after about the fourth day, by which time typical rashes or suggestive temperature readings should have manifested themselves. Occasionally an atypical salmonella infection might cause difficulty. Cases of this occurred ashore, in which pyrexia, malaise and limb pains were conspicuous, and gastro-intestinal symptoms completely absent, yet bacteriological investigation established the true diagnosis.

As regards treatment, most of the cases were given tincture of ammoniated quinine after diagnosis had been established, with A.P.C. tablets

and a linctus as required. They were largely mild cases and no controls were kept for comparison. Of the more severe cases, one was dramatically improved as regards temperature by administration of quinine, grs. v four-hourly, but physical signs and cough were not influenced. Sulphapyridine was tried on two patients, one complicated and one uncomplicated, with inconclusive results. Certainly a mixed infection by staphylococci and *H. influenzæ* would not suggest any specific reaction to sulphapyridine, and this is in accordance with reports from other sources. Hubble and Osborn, however, stress the value of the sulphanilamides in infants, but without mentioning any bacteriology.

Ætiology of Influenza Reviewed.—The term "influenza" has been, and still is, unfortunately used to describe almost any pyrexial illness to which a diagnosis cannot be given. This loose terminology denotes muddled thinking and cannot be too strongly condemned. A symptomatology exists, to which the term "influenza" has been given; well-defined complications are recognised, and typical sequelæ may delay a return to normal. Many examples of such infections are recorded in the literature; these include widespread pandemics which have swept across the world at intervals during the last one hundred years, and also smaller epidemics of more recent times.

It seems that uncomplicated influenza is a disease with a very low mortality. In this small series the death-rate was nil, and large epidemics have been reported with a similarly low figure. Thus, of 55,263 cases occurring in the German Army during one outbreak the mortality was only 0·1 per cent. On the other hand, the death-rate of the great pandemic of 1918-19 was a very high one. A search into the possible causes of this variation in mortality brings one to the much-discussed question of the ætiology of influenza.

In 1892 Pfeiffer[5] described a small Gram-negative coccobacillus which he claimed to be the causal organism of influenza. This claim was based on the frequency with which *H. influenzæ* was found in the respiratory tract secretions of patients suffering from influenza, and was supported by various clinical observations and investigations. Thus Meunier, many years ago, found the bacillus circulating in the bloodstream in four out of eight cases of typical influenza. Further evidence of a generalised bloodstream infection was forthcoming by the publication of cases of bacterial endocarditis in which the specific Pfeiffer bacillus had been cultured from the vegetations.

An obvious alternative to Pfeiffer's theory was the possibility that the organism was a normal inhabitant of the respiratory tract, and Lord, of Boston, demonstrated its presence in thirty out of one hundred unselected cases of acute and chronic bronchitis.

Finally, the bacillus might easily have been a secondary invader following closely on the heels of some primary infecting agent. That it is not an invader of any great pathogenicity is suggested by the rarity of its occurrence in pure culture in any of the complicating conditions of the chest. Thus bronchopneumonia and empyema are common complications, but in sputum cultures from the former Pfeiffer's bacillus is usually heavily overgrown by either the pneumococcus, streptococcus or staphylococcus; and in empyema *H. influenzæ* is an extreme rarity. So much for the theory of the bacterial origin of influenza, and it must be admitted that the evidence is too inconstant to justify general acceptance.

The mode of onset, symptoms, and absence of initial signs in influenza are very characteristic of virus infections in general, and in 1933 Andrewes, Smith and Laidlaw[6] published the first of a series of experiments which quickly became world-famous. These workers inoculated into ferrets the filtered throat washings from influenza patients, and produced a constant pathological state in the animals. Hence they concluded that the causative organism in man was a filter-passing virus; they further confirmed its association with influenza by measuring the antibody titre in the sera of patients in the acute pyrexial phase of the illness, and again in the convalescent stage, and demonstrated a steady rise.

This work is the basis of the present-day acceptance of the virus as the cause of influenza. But further research revealed the presence of different types of virus responsible for different epidemics. Stuart Harris, Wilson Smith and Andrewes[7] investigated a large number of epidemics in public and Service institutions during 1939, and in the great majority of cases were unable to demonstrate a virus. This contrasted sharply with the 1937 outbreaks, which were nearly all virus-positive. They also stated that they were unable to distinguish, from the clinical picture at the bedside, between the virus-positive and virus-negative cases. The former were said to be due to the virus A and the latter were presumed to be due to an unknown virus X.

Recently Francis[8] has brought to light a hitherto undescribed virus which may be similar to this X strain or may be a third variety. He isolated from various influenzal epidemics in the United States a virus which was serologically different from the A virus and which he called virus B. There seem to be, therefore, at least two distinct viruses causing two types of epidemic influenza, which are clinically indistinguishable one from the other; and these, in their turn, may on occasion be difficult to differentiate from other virus diseases such as sandfly fever, lymphocytic choriomeningitis and polioencephalitis, especially when the neurotropic element of the two latter is little in evidence, as may be the case in some patients.

This work, carried out in England and America since 1932, has proved,

by general consent, the virus ætiology of influenza, but it does not necessarily exclude Pfeiffer's bacillus from all participation. How are we, otherwise, to explain the undoubted occasional invasion of the bloodstream, and more frequent massive invasion of the lungs, by this bacillus during an attack of influenza? A reasonable explanation—and one which has been put forward by Harris, Smith and Andrewes—is that the presence of the influenza virus in the body facilitates infection by visible bacteria, which themselves give rise to complications, most often of a respiratory nature. There seems no reason why such "team work" should not exist, or why the secondary invader should not under certain conditions reach the bloodstream at an early stage of the illness, to be concentrated in one particular system at a later date. *H. influenzæ* is not an easy organism to culture, and failure to isolate it from the blood does not necessarily exclude its presence, particularly as it may very well follow the example of other bacteria such as the pneumococcus in lobar pneumonia, and the meningococcus in cerebrospinal meningitis, and circulate in the bloodstream for perhaps only a few hours at the beginning of the infection.

Pfeiffer's bacillus, under these circumstances, seems to be a fairly innocuous organism, but unfortunately it is not the only, or even the commonest, secondary invader. The pneumococcus, streptococcus and staphylococcus, in that order of frequency, are only too often co-invaders. And on them depends—and this cannot be stressed too strongly—the mortality of an influenza epidemic.

In reviewing the cases reported in this paper as they concern the bacteriology of influenza in general, two points are worthy of note. Firstly, the isolation of a pure culture of *H. influenzæ* from the blood of an otherwise uncomplicated case of typical influenza, for the presence of which a possible explanation has been advanced above; and secondly, the identification of the staphylococcus as the pathogenic secondary invader in the cases complicated by respiratory symptoms. Not only was it present in pure culture in the alveolar fluid and empyema pus of two patients, but it was also the predominating organism both in direct stained films, and in culture of sputum from almost all the other cases. Its presence, it is thought, explains the lack of toxicity in almost all patients, even in those with extensive lung signs, and also the indolent progress of many of the symptoms. The one case of empyema, apart from an initial moderate rise in temperature, showed no signs of toxicity whatsoever.

The effect of the secondary invading organism on the course and ultimate outcome of influenza was dramatically stressed shortly after this outbreak had died down by the admission to the hospital ship of a patient who had been taken ill shortly after leaving England. He apparently

developed a typical influenzal bronchitis, which progressed to a bilateral bronchopneumonia. This was soon complicated on the left by an interlobar empyema, which ruptured into the lung, producing a lung abscess, extensive empyema and bronchopleural fistula. After closed intercostal drainage had produced a temporary improvement, the patient died suddenly of a large hæmorrhage from the lung root. The invading organism was a virulent hæmolytic streptococcus. How different was the clinical picture and course of the disease compared with that produced by his sluggish colleague the staphylococcus !

Summary.

An outbreak of influenza is described. This occurred among His Majesty's forces serving on a station in the tropics.

A clinical description of the cases is given and special attention is paid to the respiratory complications.

The differential diagnosis of influenza from the commoner tropical and non-tropical diseases is discussed.

The present position of the influenza problem is briefly summarised.

Acknowledgments.

My thanks are due to Surgeon Captain Drennan, D.S.O., R.N., for permission to publish this paper; also to Surgeon Commander Wear, R.N.V.R., and Surgeon Lieutenant-Commander Murray, R.N.V.R., together with their technicians, for their respective radiological and bacteriological assistance; also to Major Maegraith, R.A.M.C., for his confirmation of the bacteriological findings.

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MEETINGS OF SOCIETIES

A MEETING of the Joint Tuberculosis Council was held on Friday, February 20, in London.

Present: Dr. D. A. Powell (Chairman), Drs. G. Lissant Cox, J. Ferguson, J. C. Gilchrist, G. T. Hebert, L. E. Houghton, G. Jessel, S. Vere Pearson, N. Lloyd Rusby, D. P. Sutherland, N. Tattersall, Professor W. H. Tytler, Dr. H. G. Trayer, Mr. J. E. H. Roberts, Drs. E. Ward, J. Watt, and Hon. Secretary (Dr. J. B. McDougall).

The question of the shortage of nurses in sanatoria was raised once more in connection with the report of the Nursing Committee, and the Secretary was instructed to communicate with the Ministry of Health and the General Nursing Council requesting that further consideration be given to the establishment of a supplementary register for tuberculosis nurses.

A Committee, with Dr. N. Tattersall as Convener, was set up to consider the question of notification of cases of tuberculosis with a view to defining the position more accurately.

The following officers were elected for 1942:

Chairman: Dr. D. A. Powell.
Vice-Chairmen: Drs. James Watt and E. Ward.
Hon. Treasurer: Dr. G. Jessel.
Hon. Auditor: Dr. D. P. Sutherland.
Hon. Secretary: Dr. J. B. McDougall.

The Hon. Auditor reported that the balance in hand at the end of 1941 was £71 15s. 1d. and that assistance for clerical work and other incidental expenses had been forthcoming from the Ministry of Health, to take effect as from April 1, 1942. The Treasurer's report was accepted and the statement of accounts passed.

A discussion on various methods for giving wider publicity to reports of the Council took place, and the matter was finally left with the officers of the Council.

The Committee which had previously been convened by Dr. Hawthorne (resigned), and which had been responsible for advice on milk, was requested to take over questions of nutrition generally, and Dr. L. E. Houghton was appointed the new Convener of this Committee. Drs. Trayer and Rusby were appointed as additional members of the same Committee.

Co-ordination of Tuberculosis Organisations.—A lengthy discussion on the question of co-ordination of the various organisations dealing with tuberculosis in this country followed, and letters were read from the National

Association for the Prevention of Tuberculosis, the Tuberculosis Association, and from the Tuberculosis Group of the Society of Medical Officers of Health.

The National Association asked that the J.T.C. should send five representatives to a conference at which the Tuberculosis Association would also be represented; the Tuberculosis Association invited the opinion of the Council on the question of co-ordination generally, and the Tuberculosis Group of the Society of Medical Officers of Health stated that it was their opinion that the time was inopportune for drastic alterations in the organisations dealing with the varied aspects of the tuberculosis problem, and also suggested that the work of the Standing Advisory Committee should be transferred to the Joint Tuberculosis Council, which, in their opinion, was fully representative of all organisations dealing with the many aspects of tuberculosis work.

An interesting discussion followed in which the majority of members took part. It was clear, however, that there was no measure of general agreement.

Drs. Sutherland, Jessel and Trayer supported the view that the Joint Tuberculosis Council was in fact the body which could deal adequately with all the problems which had to be discussed, and that the Standing Advisory Committee had undertaken much of the work which had previously been allocated to the Joint Council. Dr. G. Lissant Cox felt that much of the present difficult position was due to the fact that one organisation was ignorant of the work of the other. The view of certain members of the Tuberculosis Association was expressed by Drs. Hebert and Rusby, who said that an organisation with over three hundred members felt that the time had come for them to be more adequately represented on bodies dealing with national problems in tuberculosis.

Dr. Hebert gave some instances of gross overlapping between committees, and Dr. Vere Pearson felt that the National Association ought to take the lead in administrative matters and that the other organisations ought to be constituted sub-groups of the National Association.

It was resolved that the invitation of the National Association to send five representatives to meet members of the other organisations be acceded to, and the following were elected to attend: Dr. J. Watt, Dr. G. Lissant Cox, Dr. D. P. Sutherland, Dr. G. Jessel, and the Hon. Sec.

It was further resolved that the National Association be requested to invite a similar number of members from the Tuberculosis Group of the Society of Medical Officers of Health.

A motion by Dr. G. Jessel to invite the Ministry of Labour and National Service to send an observer to the meetings of the Joint Council was passed.

The next meeting of the Council will be held in London on Saturday, May 16, at 10 a.m.

DR. PERCY KIDD

AN APPRECIATION

DR. PERCY KIDD died on January 21 in his 91st year. He was the son of Dr. Joseph Kidd, and was educated at Uppingham and Oxford. In 1873 he graduated with first-class honours in Natural Science and entered as a medical student at St. Bartholomew's Hospital. In 1878 he became M.R.C.S. and proceeded M.A. and B.M. at Oxford. He was elected in the same year to the Radcliffe Travelling Fellowship, and went to study at Strasburg and Vienna. At St. Bartholomew's Hospital he served as house physician and later as casualty physician, assistant medical tutor and demonstrator of physiology.

In 1879 he became a Member of the Royal College of Physicians and in 1885 was elected to the Fellowship.

In 1881 he was elected to the active staff of the Brompton Hospital, and served until 1906, when he joined the consulting staff. In 1882 he proceeded D.M. at Oxford. In 1890 he was appointed assistant physician to the London Hospital, becoming full physician in 1899; he continued his service until 1920, when he joined the consulting staff.

Kidd's early training had given him wide general culture and a knowledge of contemporary medical science which proved to be of great value when he became lecturer in general pathology and in medicine at the London Hospital Medical College.

During his service at the Brompton Hospital he began to devote special attention to diseases of the heart and lungs. It was a period of active development in the knowledge of the pathology and treatment of tuberculosis.

Koch's discovery of the tubercle bacillus in 1882 and the subsequent introduction of tuberculin, the value of open-air treatment and of graduated rest and exercise, the clinical application of radiography were amongst the important innovations which affected profoundly a hospital specially devoted to the recognition and treatment of pulmonary tuberculosis.

The reputation gained by his colleagues on the staff from 1881 to 1906 gives some idea of the spirit of progress which inspired them. Theodore Williams, Frederick Roberts, T. H. Green, Mitchell Bruce, William Ewart, Kingston Fowler-Acland, Hector Mackenzie, Sidney Martin and Rickman Godlee amongst others are names inscribed in the history of medicine. Doubtless inspired by the professional staff, the Committee of Management showed great activity during the period.

The south wing of the Hospital, providing more beds and rooms for special departments, was opened in 1882. A large nurses' home came into use in 1900 and the Frimley Sanatorium was ready to receive patients in 1904.

The improved facilities offered an opportunity for the intensive clinical study of pulmonary tuberculosis of which Kidd took advantage.

He was not a prolific author; in his early years he published several papers on pathological subjects. In his clinical work he came to be recognised as one of the leaders in the specialism he had chosen. He contributed articles to Quain's *Dictionary of Medicine*, to Fowler's *Dictionary of Practical Medicine*, and to Allbutt's *Medicine*.

At the Royal College of Physicians, where he held office successively as Councillor, Censor and Senior Censor, he gave the Lumleian lecture on pneumonia in 1912 and the Harveian oration on the doctrine of consumption in Harvey's time in 1918. At the London Hospital Medical College he was lecturer on general pathology and later on medicine, but his greater love was for intimate discussion at the bedside of the cases under his care.

His quiet manner and gentle disposition did not favour the dramatic style which so often brings popularity to a lecturer, but the breadth of his views and the extent of his knowledge brought recognition of his soundness as a teacher. Both at Brompton and the London he played an important though unobtrusive part in the rapid development which occurred during his period of service. His former pupils and colleagues hold in grateful memory his kindliness, his generosity, and his wisdom.

C. W.

CORRESPONDENCE

ADVICE ON THE MANAGEMENT OF A NEW SPECIES

To the Editor, THE BRITISH JOURNAL OF TUBERCULOSIS.

"I smell a rat" (*Anon.*).

SIR,

Not only do I smell a rat, but I strongly suspect Fifth Column activity.

The letter on this subject signed so naively, "A Medical Practitioner," arouses the deepest suspicion that you have opened your correspondence columns to a subtle attack by a member of the new species against which the letter would seem to put us on our guard. This is typical of the cunning methods of invasion of this young and virile species, against which passive

resistance is worse than useless. My advice is to receive that particular specimen of the species who may have the audacity (and audacity is probably necessary) to invade your particular snug (and perhaps smug) sanatorium sanctuary, not with cold politeness, but with warm enthusiasm and open arms (and minds). Overwhelm him with work, but remind him of this: The new order came not from his thought and ingenuity, but is the natural outcome of the old. There was no place for the species in the galloping consumptive days of Queen Victoria, and it still finds no nurture in the rapid forms of tuberculosis of young races. The time-honoured régime of bed rest has played its part in thickening our fibrous capsules to withstand the pressure of collapse, and if one so young can be humble, the thoracic surgeon will admit that he is employed in correcting the exuberant efforts of nature to heal a disease that the body has already overcome.

I beg to submit myself, sir,
As yours faithfully,
A SANATORIUM SUPERINTENDENT.

REVIEWS OF NEW BOOKS

Artificial Pneumothorax in Pulmonary Tuberculosis. By T. G. HEATON. Pp. 217. The Macmillan Co. of Canada. Price $2.50.

In this book the author has successfully presented in an attractive and original manner the important subject of Artificial Pneumothorax in Pulmonary Tuberculosis. In addition to his own comments he gives a summary of the extensive literature which has evolved around the various problems attached to pneumothorax treatment, with numerous references. The book begins with an historical survey of the subject. Then follow instructive chapters on intrathoracic dynamics and the pathology and mode of action of artificial pneumothorax, and an explanation of the clinical benefit from pneumothorax.

The chapters on the indications, technique and complications are practical and complete. They contain information which would be difficult to find easily elsewhere. These chapters should be of particular interest to house physicians and others who might at any time have the care of pneumothorax patients. With the exception of a chapter on extra-pleural pneumothorax the author has not discussed the surgical procedures which are undertaken in collapse therapy. A welcome feature is the last chapter on oleothorax, where its indications and limitations are clearly set out.

Age Morphology of Primary Tubercles. By HENRY C. SWEANY, M.D., Medical Director of Research, Municipal Tuberculosis Sanitarium, Chicago, and Research Associate, Department of Physiology, University of Chicago. Springfield, Illinois: Charles C. Thomas. Price $5.00.

As the author claims, this book is in the nature of an elaborate monograph, describing in considerable detail his work in correlating the histological and anatomical changes in primary tubercles with the elements of time. In particular, the relationship of morphology to age in the later stages is stressed in giving some criteria for judging the age of a tuberculous lesion.

The first part of the book deals with the careful study of a hundred cases of tuberculosis having an accurate history of contact in which definite calcified lesions could be found. These were divided into two series: (1) contact under seven years of age; (2) contact at any time during life. The autopsies were conducted with great care by the author himself, an elaborate and careful examination of the lungs was made, large numbers of sections were taken and the histology of the tubercles carefully studied. Most of the cases are presented in full and the author has found a rough but certain parallelism between the age of primary tubercles and their morphology; and though there seems to be a slight but perceptible slowing of the changes during early evolution of the tubercle in adults it is not sufficient to impair the usefulness of the system of age prediction developed in the work.

The second part of the book deals with the practical application of the age-morphology principle. Two series of cases are again presented: (1) a miscellaneous group of indefinite contacts; (2) a group of nurses. Here the age-morphology relationship already developed is applied to the oldest tubercles found and by this method the time of contact estimated. Episodes in the histories of the respective patients during which contact might have occurred are examined and compared with the time estimate already arrived at. The author claims that this method of age analysis may prove useful in medico-legal work and in government cases following military and naval service to fix the time and source of infection; such help can only be extremely limited, as age-morphology criteria are applicable only to a limited group of case, and even then apply only when the case comes to autopsy.

It is only in the field of roentgenology that we come to a possible contribution of age-morphology relationship to the welfare of living patients. This aspect is dealt with in an interesting chapter, but unfortunately the author has found that the percentage of primary tubercles visible by X-ray is low, and even of these few show age changes at all.

The book is beautifully compiled, with many excellent microphotos and protocols, and is obviously the result of an enormous amount of work. Although too highly specialised to be of use to the student of general medicine it will have an interest to those working in the field of tuberculosis.